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THE SURGICAL TREATMENT OF GLOSSOPHARYNGEAL NEURALGIA *

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ROCHESTER, MINN.

Glossopharyngeal neuralgia is a disease similar to trifacial neuralgia in that the pains associated with it are spasmodic, excruciating and lancinating, but radiate from the pharynx and tonsillar fossa to the ear. The pain is brought on by swallowing or yawning, and lasts for only a few moments; the intermittent periods of pain and ease may continue for from a few weeks to several months, but always recur. Glossopharyngeal neuralgia simulates trifacial neuralgia, temporary relief being obtained by peripheral avulsion. In all probability the disease involves the superior jugular ganglion of the glossopharyngeal nerve, and while it may be possible occasionally to avulse both the petrosal and the jugular ganglion from an extracranial approach, the only assurance of permanent relief lies in division of the glossopharyngeal nerve proximal to the superior ganglion, through an intracranial approach.

ANATOMY

The ninth or glossopharyngeal nerve is a mixed nerve, containing motor and sensory fibers, the latter including those transmitting the impulses of the special sense of taste. The motor element is quite small and supplies only, according to Piersol, the stylopharyngeus muscle and secretory fibers to the parotid gland, while the sensory fibers are distributed to the mucous membrane of the middle ear, tonsillar fossae, tongue and oral pharynx.

Ransom discusses the glossopharyngeal nerve as a mixed nerve, made up of general and special visceral afferent fibers, and general and special visceral efferent fibers. The general and special afferent fibers have their cells of origin in the petrous ganglion; their central branches terminate in the solitary tract and its nucleus. The general viscero-efferent fibers arise from the cells of the inferior salivatory nucleus of the medulla oblongata; the special viscero-afferent fibers arise in the

* From the Section on Neurologic Surgery, Mayo Clinic,

* Read before the Section on Nervous and Mental Diseases at the Seventy-Fifth Annual Session of the American Medical Association, Chicago, June, 1924.

nucleus ambiguus of the medullary continuation of the dorsal lateral cell group of the anterior column of the cord.[1]

Superficially, the glossopharyngeal nerve takes its origin from the rostral end of the posterolateral sulcus of the medulla oblongata in a line with the tenth and eleventh nerves. Like the vagus, the glossopharyngeal nerve has two ganglions: the dorsal root ganglion is at the upper orifice of the jugular foramen; the trunk or petrous ganglion is in a small depression at the lower orifice of the jugular foramen. From the latter, branches of communication pass to the auricular branch of the vagus, to the jugular ganglion and to the sympathetic nerve.[2]

As the glossopharyngeal nerve traverses the jugular foramen, it lies anterior to the tenth and eleventh nerves in its own separate dural sheath. It occupies a groove, or sometimes a bony canal, in the foramen. Emerging from the foramen the nerve passes between the internal carotid artery and the internal jugular vein, and dipping beneath the styloid process follows a downward course along the posterior border of the stylopharyngeus muscle, with which it passes between the internal and the external carotid arteries. Turning gradually forward it reaches the outer side of the stylopharyngeus muscle and stylohyoid ligament, and disappears beneath the hyoglossus muscle to break up into its terminal branches to the tongue.[3]

The branches of the glossopharyngeal nerve are: (1) tympanic, (2) pharyngeal, (3) muscular, (4) tonsillar and (5) lingual.

1. The tympanic nerve *(n. tympanicus)*, or Jacobson's nerve, arises from the petrous ganglion as its most important branch and traverses a tiny canal in the osseous bridge between the jugular fossa and the carotid canal. Entering the tympanic cavity and receiving fibers from the carotid plexus of the sympathetic by way of the small deep petrosal *(n. caroticotympanicus)* the tympanic nerve passes upward and forward in a groove on the promontory, and breaks up in this situation to form the tympanic plexus (plexus tympanicus Jacobsoni). After distributing filaments to the mucous membrane lining the tympanic cavity and the associated air-spaces (mastoid cells and eustachian tube), its fibers reassemble and join with a filament from the geniculate ganglion to continue as the small superficial petrosal nerve to the otic ganglion.

Branches.—These are: (*a*) the small superficial petrosal nerve, (*b*) the branch to the fenestra ovalis, (*c*) the branch to the fenestra rotunda, (*d*) the branch to the eustachian tube, (*e*) the branch to the mastoid cells and (*f*) the branch to the great superficial petrosal nerve.

(*a*) The small superficial petrosal nerve (*n. petrosus superficialis minor*) is the continuation of the tympanic nerve, formed by a reassembling of the fibers of the plexus, supplemented by a filament from the geniculate ganglion of the facial. It traverses a canal which begins at the anterior superior portion of the tympanic cavity, passes beneath the upper end of the canal for the tensor tympani

1. Ranson, S. W.: The Anatomy of the Nervous System from the Standpoint of Development and Function, Philadelphia, W. B. Saunders Company, 1921.

2. Piersol, G. A.: Human Anatomy, Ed. 5, Philadelphia, J. B. Lippincott Company, 1916, p. 1263.

3. Piersol, G. A.: Human Anatomy, pp. 1264-1265.

and appears on the superior surface of the petrous portion of the temporal bone, to the outer side of the cranial opening of the hiatus Fallopii. While in the canal, it sometimes receives a communicating branch from the great superficial petrosal nerve. It leaves the cranium through a canal in the greater wing of the sphenoid, or through the fissure between the greater wing and the petrous portion of the temporal bone, and on reaching the base of the skull, joins the otic ganglion as its sensory root.

(*b*) The branch to the fenestra ovalis supplies the mucous membrane in the neighborhood of the oval window.

(*c*) The branch to the fenestra rotunda is distributed to the mucous membrane over and around the fenestra.

(*d*) The branch to the eustachian tube supplies the mucous membrane, lining the osseous portion of that canal.

(*e*) The branch to the mastoid cells supplies the mucous lining of these cells.

(*f*) The branch to the great superficial petrosal nerve joins the latter in the hiatus Fallopii.

2. The pharyngeal branches (*rami pharyngei*) number two or more, of which the largest descends along the course of the internal carotid artery and joins the pharyngeal branches of the vagus and sympathetic to form the pharyngeal plexus, which supplies the mucous membrane and muscles of the pharynx. The smaller pharyngeal branches pierce the superior constrictor and are distributed to the mucous membrane lining the upper portion of the pharynx.

3. The muscular branch (*ramus stylopharyngeus*) enters the stylopharyngeus, and, after giving off fibers for the supply of that muscle, passes through it to be distributed to the mucous membrane of the pharynx.

4. The tonsillar branches (*rami tonsillares*) are given off near the base of the tongue. They are slender filaments which form a plexiform ramification, the circular tonsillaris, around the tonsil. From this plexus filaments are distributed to the tonsil, the soft palate and the faucial pillars.

5. The lingual branches (*rami linguales*) are the two terminal filaments of the nerve. The larger posterior branch passes upward and separates into a number of filaments which supply the circumvallate papillae and the mucous membrane covering the posterior part of the dorsum of the tongue, the glossoepiglottic and pharyngo-epiglottic folds and the lingual surface of the epiglottis. The smaller anterior branch supplies the mucous membrane of the side of the tongue half way to the tip.[3]

REVIEW OF LITERATURE

Doyle,[4] in his study of glossopharyngeal neuralgia, reports four cases, and emphasizes the diagnostic symptoms, besides making special studies of the sensory and gustatory changes following avulsion of the glossopharyngeal nerve.

In the first of this series of four cases, the patient came to the Clinic complaining of paroxysms of pain in his throat in the region of the tonsillar fossa with radiation to the ear, brought on by swallowing and chewing. He was regarded as having atypical trifacial neuralgia,

4. Doyle, J. B.: A Study of Four Cases of Glossopharyngeal Neuralgia, Arch. Neurol. & Psychiat. **9**:34-46, 1923.

but as he had had trouble for a number of years and had experienced no relief from temporary measures, division of the posterior sensory root of the gasserian ganglion on the right side was advised. Unfortunately, this did not relieve the pain. The patient was again examined, and his case especially studied by Doyle and Greenfield; the following findings were submitted: The anesthesia, after division of the sensory root of the gasserian ganglion on the right side, was complete for pain, temperature and touch over the area of the cutaneous distribution of the right trigeminal nerve, and there was complete anesthesia over the mucous membrane of the right half of the buccal cavity, the right half of the alveolar processes, the tongue and hard palate, and the anterior half of the right nasal cavity; touch and pain senses, however, were normal over the entire surface of both external auditory canals, the external aspect of both tympanic membranes and both auricles. Following extracranial avulsion of the glossopharyngeal nerve and the pharyngeal branch of the vagus, the patient complained of a sense of fulness in the right side of the pharynx with slight dysphagia, and had complete anesthesia to pain, temperature and tactile sensation over the entire right half of the inferior aspect of the soft palate. On its upper aspect, the right half of the soft palate was completely anesthetic for 1.5 cm. anterior to its posterior border; all of the portion of the right half of the pharynx lying between the level of the hard palate above and the pyriform fossa below, was completely anesthetic to pain, tactile and temperature stimulation; pain, temperature, and touch were normally appreciated over the posterior half of the nasal cavity, and over all of the nasal pharynx above the level of a line drawn through the hard palate. Sensation was normal over the epiglottis and the upper aspect of the larynx; taste was absent for salt, sweet, bitter and sour over the entire right half of the tongue. The right half of the soft palate and the right superior, middle, and inferior constrictors were totally paralyzed, with some loss of tone, but without actual loss of power, on the right half of the tongue.

In Doyle's discussion of the sensory and gustatory changes, he refers to the work of Lucianni, Carl, von Urbantschitsch and Panizza, who believe that the taste fibers are derived entirely from the ninth nerve, and that the chorda tympani is composed of secretory and vasodilator fibers only, which correspond to the findings in Doyle's Case 1 of the series reported. In investigating the sensory distribution of the trigeminal nerve, he studied other patients following the division of the sensory root for trifacial neuralgia, and the findings were revealed to be the same as those in Case 1 before the avulsion of the glossopharyngeal nerve. With reference to the sensory innervation, he refers to the work of Schafer and Symington, who believe that the sphenopalatine ganglion derives sensory fibers from the maxillary and glossopharyngeal nerves,

and from the geniculate ganglion of the seventh nerve with only a few fibers coming from the maxillary nerve, most of these being distributed to the nose and palate. The nerve to the nasopharynx originates in the back of the ganglion, often in common with the vidian nerve.

Doyle says, in consideration of these data, that it is obvious that the nasopharynx and a variable portion of the posterior part of the nasal cavity receive their afferent fibers from a source other than the trigeminal nerve, and the trunk of the ninth. At the time of operation the glossopharyngeal nerve was avulsed from the jugular foramen. The actual rupture of the nerve occurred at the lower end of the petrous ganglion. For this reason it cannot be determined with any degree of certainty whether or not the tympanic branch of the ninth nerve was interrupted in its course. This leaves the question open as to whether the afferent fibers of the sphenopalatine ganglion concerned with innervation of the nasopharynx are derived from the intermediate nerve through the geniculate ganglion, the greater superficial petrosal nerve and the nerve to the pterygoid canal, or from the small branch from the tympanic plexus to the greater superficial petrosal; hence the sensory distribution of the nasopharynx can be determined only after an intracranial supraganglionic division of the glossopharyngeal nerve.

In 1918, Vernet,[5] in discussing the syndrome of the jugular foramen, said that a destructive lesion of the ninth nerve causes paralysis of the superior constrictor of the pharynx, which results in difficulty in swallowing solid food. Paralysis of the tenth nerve results in anesthesia of the soft palate and the posterior wall of the pharynx. Destruction of the accessory part of the eleventh nerve leads to paralysis of the soft palate and larynx, while destruction of the spinal portion produces paralysis of the sternocleidomastoid and trapezius muscles.

In 1907 and 1908, Hunt[6] discussed in detail inflammations of a herpetic nature involving the ear and adjacent regions. He attributed them to an involvement of the geniculate ganglion similar to involvement of the gasserian ganglion and dorsal root ganglions in herpes zoster. The pain was different from that typical of tic douloureux.

In 1908, Orbison[7] reported a case of herpes of the tympanic membrane, and endeavored to prove the condition to be "due to zosteroid affection of the petrosal ganglion."

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5. Vernet, M.: Syndrome du trou déchiré postérieur (Paralysie des nerfs glossopharyngeal, pneumogastrique, spinal), Rev. neurol. **34**:117-148, 1918.

6. Hunt, J. R.: A Further Contribution to Herpetic Inflammations of the Geniculate Ganglion: A Syndrome Characterized by Herpes Zoster Oticus, Facialis, or Occipitocollaris, with Facial Palsy and Auditory Symptoms, Am. J. M. Sc. **136**:226-241, 1908.

7. Orbison, T. J.: Herpes of the Membrana Tympani Due to Zosteroid Affection of the Petrosal Ganglion, J. Nerv. & Ment. Dis. **35**:500-506, 1908.

In 1910, Mills [8] refuted Hunt's arguments concerning the nature of the afferent fibers of the facial nerve, and attempted to show that the function of the nerve was chiefly gustatory, and that only a vestigial remnant of common sensation remained over a small portion of the anterior part of the tongue, and a narrow strip in the auricle.

Lesions of the glossopharyngeal nerve, experimental or acquired, are extremely rare. In 1896, Meyer [9] described the changes in the taste buds of foliate type of the tongue of the guinea-pig subsequent to section of the ninth nerve.

In 1919, Neve [10] described a case of what he termed herpes zoster of the glossopharyngeal nerve. The patient had peripheral facial nerve palsy on the left, impairment of the eighth nerve, vesicular eruption over the left half of the soft palate and pain posterior and inferior to the left ear and down the left side of the neck.

Oppenheim [11] described a case of paralysis of the ninth nerve in a woman. Objectively she had a thermesthesia, paralysis of the soft palate and pharyngeal muscles, and loss of taste in the soft palate, pharynx and posterior part of the tongue. Tactile sense was preserved intact.

In 1910, Weisenburg [12] described a case of cerebellopontile tumor, diagnosed for six years as tic douloureux. Frazier cut the sensory root of the fifth nerve and excised most of the gasserian ganglion relatively early, to relieve what appeared to be trifacial neuralgia. Following this operation the patient complained of pain in the right side of the throat radiating into the ear and neck, and occasionally to the shoulder, and later he had severe pain at the root of the tongue and in the right side of the pharynx. This pain was paroxysmal and was set up by the contact of food or other foreign material with the mucous membrane of the pharynx. He also complained of pain in the lower right side of the face. It should be emphasized that the character of the pain differed after the ganglion operation; before operation the pain was in the whole face as well as in the throat. Repeated pharyngeal and laryngeal exam-

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8. Mills, C. K.: The Sensory Functions Attributed to the Seventh Nerve, J. Nerv. & Ment. Dis. **37**:273-284 and 354-379, 1910.

9. Meyer, S.: Durchschneidungs-Versuche am Nervus Glosso-pharyngeus, Arch. f. mikr. Anat. **48**:143-145, 1896.

10. Neve, C. T.: Herpes Zoster of the Glossopharyngeal Nerve, Brit. M. J. **2**:630, 1919.

11. Oppenheim, H., quoted by Weisenburg, J. A. M. A. **54**:1602 (May 14) 1910.

12. Weisenburg, T. H.: Cerebello-Pontile Tumor Diagnosed for Six Years as Tic Douloureux: The Symptoms of Irritation of the Ninth and Twelfth Cranial Nerves, J. A. M. A. **54**:1600-1604, 1910.

inations revealed excessive tenderness on the right side, but no weakness of any of the muscles of that region. At necropsy a tumor was discovered in the right cerebellopontile angle, lying directly on the sensory and motor roots of the fifth nerve. The ninth and tenth nerves, which were prominent, stretched over the lower portion of the tumor. On account of the close relationship of the peripheral fibers of the fifth and ninth nerves, Weisenburg interpreted the pain, which persisted in the area of distribution of the fifth nerve after resection of the major portion of the gasserian ganglion, as a result of irritation of the ninth nerve. The pharyngeal pain was also attributed to irritation of the ninth nerve.

In 1920, Sicard and Robineau [13] described three cases of what they termed "algie velo-pharyngée essentielle." The first two cases were seen in the French army in 1916 and 1917, respectively. In one case, the pain was right sided; in the other, left. In neither case could a cause be established. There was no antecedent history of syphilis, and the condition did not respond to treatment for syphilis, to sedatives or physical agents. In both cases the pain had persisted over a period of several years; it was not continuous, but recurred frequently day and night in extremely acute paroxysms on the least movement of mastication, deglutition or speech. Occasionally the paroxysms occurred spontaneously. The patients developed suicidal tendencies on account of the apparent incurability of the condition. The treatment advised consisted in section of the glossopharyngeal nerve and the pharyngeal branches of the vagus, with ablation of the superior cervical sympathetic ganglion. These two patients were operated on by surgeons unfamiliar with surgical procedures in the neck. In one the ninth nerve was cut, the sympathetic ganglion was removed, and the trunk of the vagus severed; in the other, the ninth and twelfth nerves and the pharyngeal branches of the tenth nerve were cut, and the sympathetic ganglion was removed. Both patients convalesced rapidly. The first patient developed unilateral palsy of the vocal cord; the second, hemiatrophy of the tongue. The third patient was a woman, aged 50, in whom Sicard successfully cut the ninth nerve and the pharyngeal branches of the tenth, and ablated the superior cervical sympathetic ganglion. During operation the trunk of the vagus was pulled, resulting in paralysis of the homolateral vocal cord; this condition, however, was steadily improving. As in the first two instances, all pain disappeared immediately following operation. The first patient has been completely relieved for four years and the second for three.

13. Sicard, R., and Robineau: Communications et presentations. I. Algie vélo-pharyngée essentielle. Traitement chirurgical, Rev. neurol. **36**:256-257, 1920.

Harris [14] has recently described two cases of a rare form of chronic paroxysmal neuralgia, which he termed glossopharyngeal neuralgia. The pain, according to him, characteristically starts in the throat in the region of the tonsil and anterior pillar and radiates to the ear and upper part of the neck. His first patient was a man, aged 40, whom he had first seen ten years before, when he successfully injected the third division on the affected side without influencing the pain. His second patient was a woman, aged 87, who had suffered from the condition for ten years. Harris was convinced of the involvement of the ninth nerve in these cases; he had seen a man with similar pains and intense hyperesthesia of the side of the neck following a recurrence of epithelioma of the tonsil seven years after extirpation of a malignant vocal cord.

SYMPTOMS

In 1910, Weisenburg described the glossopharyngeal nerve syndromes, and in 1920, Sicard and Robineau reported three cases with definite glossopharyngeal neuralgia. Harris, in 1921, described two cases of typical glossopharyngeal neuralgia. In 1923, Doyle, of the Mayo Clinic, described four cases, and since then five cases have been added to the Mayo Clinic series; in four of the cases, operation has been performed. In three cases, the glossopharyngeal nerve was avulsed peripherally, and in one case the pharyngeal branch of the tenth nerve was avulsed, being mistaken for the ninth cranial nerve.

The disease simulates trifacial neuralgia in its occurrence, duration, repetition, and the character of pain, but differs from trifacial neuralgia in its distribution and has a trigger zone in the tonsillar fossa. It is brought on more often by swallowing than by chewing, and it cannot be brought on by rubbing the face and only occasionally by rubbing the ear. Glossopharyngeal neuralgia also differs from trifacial neuralgia in that the pain radiates from the pharynx and tonsillar area through the neck to the ear, particularly to the tympanum. In cases in which there is any doubt concerning a diagnosis of glossopharyngeal neuralgia, it will be found that it is difficult to bring on paroxysms of pain by stimulating the pharynx, if the pharynx and tonsillar area have been thoroughly cocainized, which will not inhibit paroxysms of pain in the maxillary and mandibular branches of the trigeminal nerve in trifacial neuralgia. Glossopharyngeal neuralgia also is unlike trifacial neuralgia, since it is impossible to inject the nerve with alcohol on account of its close proximity to the vagus, hypoglossal and spinal accessory nerves, but temporary relief can be obtained by peripheral avulsion just as this measure affords temporary relief in trifacial neuralgia. However, since

14. Harris, W.: Persistent Pain in Lesions of the Peripheral and Central Nervous System, Brain **44**:557-571, 1921.

the peripheral avulsion is such a formidable procedure, I recommend the intracranial division, even though I have not performed this operation except on a cadaver. My investigations on the cadaver convince me that the procedure can be undertaken with greater ease than the surgical removal of an acusticus tumor.

PERIPHERAL AVULSION

In my first endeavor to relieve glossopharyngeal neuralgia surgically, I had hoped to be able to avulse the petrosal and superior ganglions by an extracranial route. The following technic was used: The incision was made parallel to the anterior border of the sternocleidomastoid muscle, extending upward for a distance of 2.5 cm. above the tip of the mastoid, just behind the ear, then downward from the tip of the mastoid for a distance of 10 cm. On exposing the sternocleidomastoid, it was found to be advantageous to divide half of its attachment on the mastoid and retract it outward, in order to obtain sufficient exposure; the dissection was then carried inward and upward behind the angle of the mandible along the skull, dividing the posterior belly of the digastric and stylohyoid muscles, gently retracting the parotid upward and outward. On retraction of the parotid, the spinal accessory was the first nerve exposed; this passes obliquely downward and outward from the jugular foramen underneath the posterior belly of the digastric. In order to obtain greater exposure, the external carotid was divided after the occipital artery had been given off, and the hypoglossal nerve, which lies superficial and lateral to the jugular vein, was exposed. On dissecting free the jugular vein, the vagus nerve was brought into view, with a mesial pharyngeal branch which might be mistaken for the glossopharyngeal nerve. By carrying the dissection back to the jugular foramen, it can be identified as a part of the vagus nerve; carrying the dissection mesially, the internal carotid, and then the upper portion of the cervical sympathetic ganglion are encountered. After the vagus, the internal carotid, and the sympathetic nerves have been identified, the glossopharyngeal nerve will be found emerging from the jugular foramen, anterior to the tenth nerve, crossing anterior to the internal carotid, dipping beneath the styloid process, following a downward course along the posterior belly of the stylopharyngeus muscle before it disappears beneath the hyoglossis muscle (Figs. 1, 2 and 3).

The foregoing technic sounds somewhat formidable; it is a delicate procedure necessitating gentle retraction of nerves, but it affords the patient relief, and if the ganglia with the glossopharyngeal nerve are successfully avulsed, permanent relief may be obtained. On closure of the wound, the divided muscles are sutured with mattress sutures. The postoperative care of the patient is that usually given in general surgical cases.

REPORT OF CASES

CASE 1.—A farmer, aged 64 years, came to the Clinic, March 16, 1922, complaining of pain in the throat and right ear. In 1912, he had had herpes zoster just above the left iliac crest. Five years before his admission to the Clinic, he had had a sharp paroxysm of pain in the region of the right ear, associated with excessive tenderness in the right auricle, immediately after taking a drink of cold water. Similar attacks occurred sporadically until 1918, when his tonsils were removed; this relieved him for about three years, when mild paroxysms began and lasted for six months. Relief was again obtained after the extraction of several infected teeth. Late in February, 1922, pain again recurred while the patient was drinking water. At the time he presented himself at the Clinic, he had had great difficulty in obtaining sufficient nourishment, owing to the pain

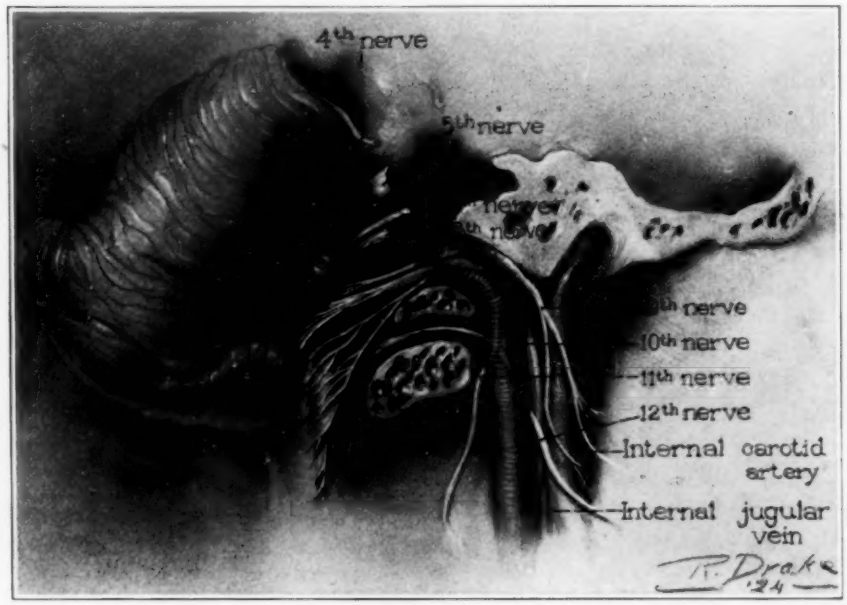

Fig. 1.—Relation of structures in jugular foramen.

induced by drinking and mastication. The pain was paroxysmal, sharp and agonizing, arising in the right fossal region and radiating laterally to the area anterior and posterior to the right ear, and lasting from thirty to ninety seconds. The frequency of the pain was definitely increased by talking and chewing. At the onset, the attacks occurred during the night; during the two weeks preceding his examination, his distress had been chiefly diurnal.

The physical examination was negative except for slight arteriosclerosis and moderate enlargement of the prostate. The systolic blood pressure was 118, and the diastolic 80. Vision was 6/6, with correction, in both eyes; both pupils were slightly irregular; the reflexes and fields were normal; both fundi were negative except for slight choroidal sclerosis; a few lenticular opacities were noted. The Wassermann reaction was negative, as were also the urinalyses, blood counts and stereoscopic roentgenograms of the head. Examination of the larynx and

nasopharynx revealed a trigger area deep in the hypopharyngeal region. The only neurologic findings were a slight diminution in perception of vibration and slight incoordination of both lower extremities.

In view of my inexperience with glossopharyngeal neuralgia at the time of this patient's examination, I felt that he probably had trifacial neuralgia of the right mandibular division with involvement of the auriculo-temple, and in view of the fact that the patient had had pain for a number of years, I advised division of the posterior sensory root of the right gasserian ganglion. This operation was performed, March 23, 1922. Immediately after the operation, the patient experienced complete relief from pain, and this lasted until April 12, when all symptoms suddenly recurred as he was yawning after waking from sleep. We

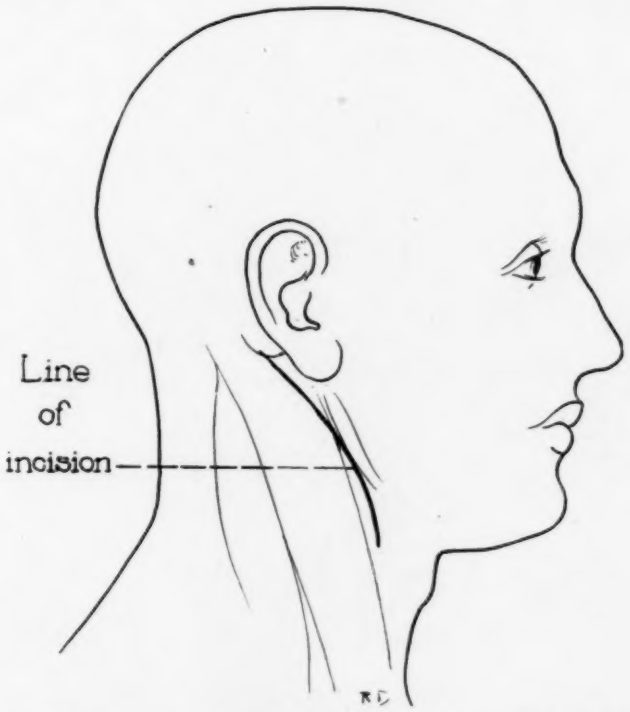

Fig. 2.—Incision for peripheral avulsion of the glossopharyngeal nerve.

then became interested in the subject of glossopharyngeal neuralgia, and Doyle and Greenfield of our staff made a special diagnostic study of the disease. The subsequent history and neurologic findings in Case 1 have been given in a discussion of Doyle's study of glossopharyngeal neuralgia. This patient is the first, and the only one of the four patients reported by Doyle, who had been operated on at that time. When last seen, May 1, 1924, he was entirely free from pain.

Case 2.—A farmer, aged 61 years, came to the clinic, April 14, 1922, because of paroxysmal pain in the region of the lobule of the left ear, and in the area of the angle posterior to the left mandible. The first attack had occurred nine years before, and had lasted from eight to ten days. The onset was insidious, with slight twinges of pain, gradually increasing in severity to the point where

morphin alone gave relief. Within from thirty-six to forty-eight hours after the onset, the pain was excruciating. The paroxysms had never occurred in series of more than two or three, and during the first attack single paroxysms had occurred at intervals of from ten to thirty minutes. The first attack subsided with the paroxysms gradually decreasing in frequency and severity until they

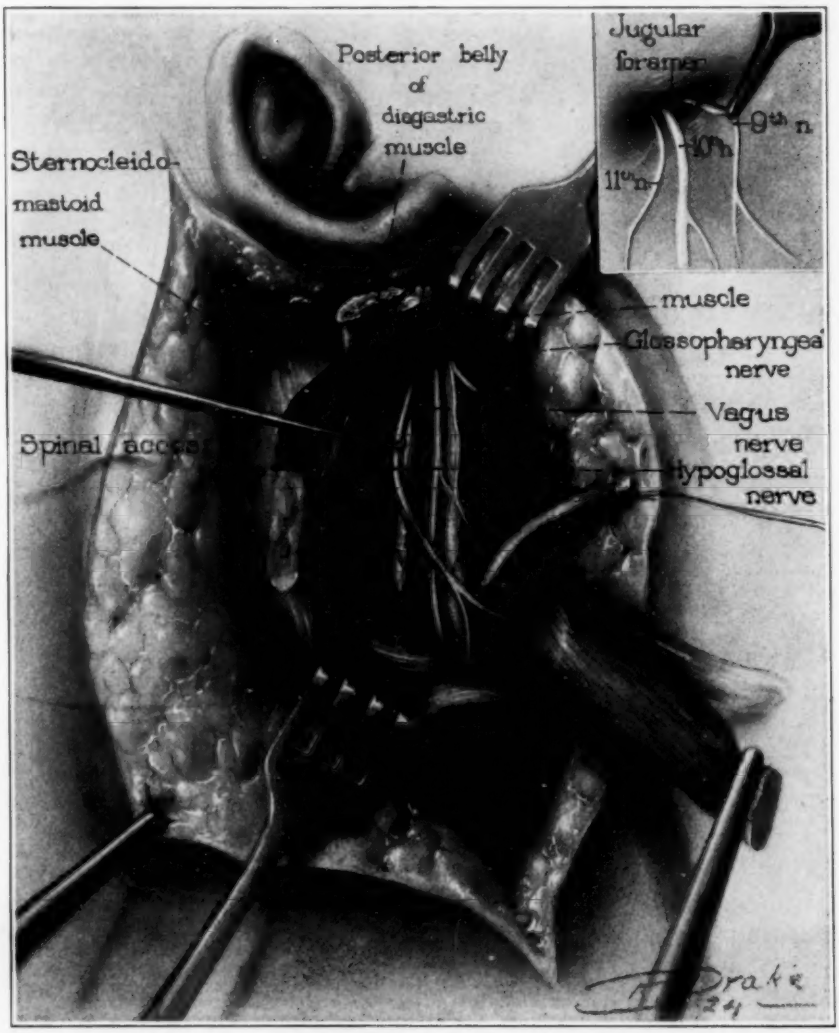

Fig. 3.—Peripheral avulsion of glossopharyngeal nerve.

disappeared and remained quiescent for three years. He then had a second attack similar to the first, lasting ten days and requiring hospitalization for seven days; morphin was again the only drug that afforded relief. The patient was free from pain for two years, when the third attack came on and remained for three weeks, with an occasional day of relief; the paroxysms were not quite so

severe as those of the first two attacks. After three years and nine months, January, 1922, the fourth attack began. This attack lasted only two weeks, and the paroxysms were severe and frequent; extraction of a tooth failed to afford relief. The patient came to the Clinic during a quiescent period.

A general examination was made, but no attempt at treatment. His last attack began on the evening of May 21, 1922; the paroxysms were very severe and were brought on by sudden movements, talking, swallowing, and drinking; fright seemed to induce some of the attacks. An attack would occasionally occur without any apparent movement of the throat or mandible; no definite trigger zone was present.

On physical examination the patient's weight was 129 pounds (57 kg.). The systolic blood pressure was 220, the diastolic pressure 110, the pulse rate 72, and the temperature normal. The heart was slightly enlarged; the pulse was irregular, and there were many extra systoles. There was evidence of moderate arteriosclerosis and slight bronchitis. Urinalysis showed an output of 1100 c.c. in twelve hours; the specific gravity was 1.020; there was a faint trace of albumin and pus, and the combined phenolsulphonephthalein test of two hours showed a return of 55 per cent. The blood and sputum were normal. The eye showed evidence of arteriosclerosis. Examination of the ear revealed a chronic otitis media with polypi in the external auditory canal. Roentgen-ray examination of the chest revealed a healed tuberculosis in the right apex. Neurologic examination revealed a slight increase in the deep reflexes, and slight incoordination. During this examination the patient was seen in an attack which came on suddenly; he was observed to seize the region about the angle of the left jaw and cough repeatedly in a hacking manner every second or two during the attack, which lasted about thirty seconds. He said that he generally coughed when the attacks were coming on, but that he coughed less during the seizure, and that he was compelled to cough occasionally between the attacks on account of a sense of irritation in the left lateral region of the throat. There seemed to be evidence of glossopharyngeal neuralgia, in addition to arteriosclerosis of the central nervous system with hypertension.

The patient was not a good surgical risk, but on account of the severe paroxysms of pain, he was anxious that operation be attempted. Avulsion of the glossopharyngeal nerve through the extracranial route was advised, and May 30, 1922, this was done with the use of combined anesthesia. In this case, I did not divide the posterior belly of the digastric and stylohyoid and the external carotid in order to obtain exposure, but approached at a lower angle than in Case 1. On account of poor exposure, greater retraction was necessary; this resulted in bleeding which clouded the field, and, to my chagrin, following the operation, I found that I had not avulsed the glossopharyngeal nerve. The pain continued, although not so severe as before, and on account of the patient's hypertension and arteriosclerosis, I did not wish to attempt an intracranial division. The patient is still having attacks.

Case 3.—A laborer, aged 52 years, presented himself at the Clinic, Nov. 4, 1922, complaining of sharp, stabbing pains in the ear, of four years' duration. At the onset of the attack, after swallowing a drink of brandy, the pains were sharp and jabbing, radiating from the right tonsil to the right ear. The first attack subsided, and the patient was free from pain for one year, when a second attack was brought on by swallowing and coughing; this lasted for two weeks. There was a period of quiescence again for from eight to ten months, when the

third attack appeared and lasted three months. All of the attacks were associated with sharp, knife-like pain, first piercing the right tonsil region and then radiating to the right ear; there was no radiation of pain elsewhere. The attacks were always brought on either by drinking cold water, or by swallowing food, talking, coughing, or turning over in bed quickly; the patient said that the pain was also brought on during the period of an attack if anything touched the right side of the throat. He said that he felt as though he wanted to clear his throat frequently, but that if he did, the seizure of pain would follow; occasionally washing the ear would bring on the pain. He gave a history of having had brain fever at the age of 4, following scarlet fever and measles, which left him with a partial facial paralysis of the left side, and impaired hearing in the left ear. Fourteen years before, he had developed otitis media in the right ear and a right mastoid abscess; this was operated on and affected the hearing on the right side.

Physical examination revealed the patient's weight to be 177 pounds (80 kg.); the systolic blood pressure was 114, and the diastolic 80; the heart and lungs were normal. Varicose veins of the left leg, a chronic ulcer on the left great toe, bilateral bunions, and left facial paralysis were the noteworthy general physical findings. The urine and blood were normal, and the Wassermann reaction was negative. Vision was 6/6 in the right eye, and 6/10 in the left; the pupils, reflexes, fields and fundus were normal; there was a ptosis of the left upper lid and weakness of the orbicular palpebrarum. Roentgen-ray examination of the chest was negative. The right membrana tympanum was entirely gone; there were granulations on the inner tympanic wall, and the mucosa was injected; the left membrana tympanum was normal; there was impairment of hearing on both sides. Neurologically, the patient was normal except for a partial paralysis of the seventh nerve on the left, the impaired hearing of both ears, and disturbed sensations of taste on both sides of the tongue, as follows: for sugar, on the anterior part of the tongue, minus 4, both right and left; on the posterior part of the tongue, minus 1 on the right and minus 2 on the left; for salt, on the anterior part of the tongue, minus 4 on the right and minus 4 on the left, but normal on both sides on the posterior portion of the tongue; for sour taste, the sensation on the anterior part of the tongue was minus 4 on the right, minus 3 on the left and on the posterior part of the tongue, normal on the right, and minus 3 on the left; for bitter taste, on the anterior part of the tongue it was minus 3 on the right, and normal on the left, and on the posterior portion of the tongue, minus 4 on both right and left. The trigger zone was located on the right posterior pharyngeal wall, adjacent to the tonsil, with no area of anesthesia in the pharynx, nasopharynx, nose, cheeks or tongue. The patient appeared to have glossopharyngeal neuralgia of the right ninth nerve, and I advised avulsion of the glossopharyngeal nerve.

On Nov. 13, 1922, the glossopharyngeal nerve was avulsed, but dissected free from the pharyngeal and laryngeal branches of the vagus, which were not avulsed. This dissection was made in order to make sure of recognizing the glossopharyngeal nerve, but apparently the dissection produced sufficient trauma to the pharyngeal and laryngeal branches of the vagus to cause temporary difficulty in swallowing and slight hoarseness which lasted for three days, but disappeared at the end of ten days when the patient was dismissed from the hospital.

The patient was completely relieved of the pain after avulsion of the glossopharyngeal nerve, and there were no paroxyms attending swallowing, chewing,

or talking. Since his dismissal the patient has worked in the woods near Fairfax, Manitoba, and no reply was received to a letter mailed to him, March 17, 1924.

CASE 4.—A railroad clerk, aged 38, came to the Clinic, Nov. 1, 1922, because of shooting pains in the region of the left tonsil, radiating to the ear and neck, posterior to the angle of the jaw on the left side. The onset of the trouble had occurred seventeen years before, while the patient was attending a football game. After cheering and rooting, he was suddenly seized with a painful spasm of the muscles of the throat; the pains were knifelike in character and radiated to the left ear. With each spasm of pain there was marked lacrimation, and he was unable to cheer during the remainder of the game; each attempt to do so precipitated excruciating pain. For the next ten days, he was unable to do any loud talking, to cough, drink cold water, or to breathe cold air without bringing on the paroxysms. The pain could also be brought on by pressure to the outer surface of the maxilla; it lasted from a few seconds to several minutes. Following this attack, the patient was free from pain for two years, when he again had a recurrence; this attack lasted for two or three days and subsided for a period of six months. Then for twelve years, he continued to have such attacks every spring and fall. On one occasion, an electric needle had been inserted in the region of the tonsils, which apparently resulted in cessation of pain for a time. Three years before coming to the Clinic, an attack more severe than the previous ones had occurred, and the pain remained more or less continuous for one year, when tonsillectomy was performed. Following this, he was relieved for six months, then had another attack lasting for fourteen days, and was again free from the pain for six months. The next attack occurred in January, 1922, and lasted until Oct. 20, 1922, when the pains gradually subsided, and at the time of examination he was not having severe pain. A dull headache accompanied the excruciating pains, and became very sharp when he stooped; the headache seemed to be most severe back of the eye, and there was frequent pain and burning of the eye during the paroxysms. The patient complained of having had three severe sick headaches during the three years previous to his examination; these, however, were not associated with neuralgic pain. He had learned that pressure on the neck posterior to the left angle of the maxilla would abort mild attacks, but would make the severe attacks worse. He complained of being irritable and grouchy during the period of attacks, and said that the moving of the jaw or any exertion of the throat would bring on the sharp flicker of pain, radiating to the eye as well as to the ear, and causing lacrimation.

Since the patient had had no pain for two weeks, we did not advise surgical treatment at this time; he was dismissed, but returned April 11, 1923. He had had another attack, starting Feb. 19, 1923, following a severe cold. The pain now radiated from the left side of the throat to the ear, centering deep in the ear, and to the tragus. From February until his second visit to the Clinic, the pain had been more or less continuous with only short periods of relief, which were brought on by yawning, sneezing, eating, inhaling a breath of cold air, and drinking cold water, especially when the water touched the throat. He described the pain as being exceedingly severe, stinging in character, and always starting in the throat, in the tonsillar region, producing an irritation which caused a desire to clear the throat; this he did constantly during an attack, with a resultant hacking cough. On a few occasions, he had been able to bring on an attack of pain by rubbing the skin back of the auricle.

The patient was well developed, 6 feet tall, and weighed 222 pounds (100 kg.). His systolic blood pressure was 120, and the diastolic 80; the pulse and temperature were normal. The blood and urine were normal, and the Wassermann reaction was negative. Vision was 6/6 in the right eye and 6/4 in the left; the pupils, reflexes, fields and fundi were normal. One tooth possibly was diseased, and a mild degree of periapical infection existed. Roentgen-ray examinations of the head and of the chest were negative. The neurologic findings were negative throughout, with the exception of the history of the glossopharyngeal nerve. There were no areas of anesthesia and no definite trigger zones, but irritation of the left tonsillar fossa would precipitate an attack, as would swallowing or yawning, and during the paroxysms the patient coughed violently in an attempt to clear the throat. Examination for taste revealed normal sensation on both sides of the tongue, anteriorly and posteriorly, for sweet, sour and bitter, and normal sensation over both sides of the posterior half of the tongue for salt, but absence of taste for salt over the anterior half of the tongue, bilaterally. In view of the history and our findings, a diagnosis of glossopharyngeal neuralgia was made, and avulsion of the glossopharyngeal nerve was advised; the three attacks of headache were believed to be of a migrainous character.

On April 16, 1923, the glossopharyngeal nerve and the superior pharyngeal branch of the vagus were avulsed. The patient's convalescence was uneventful, and he was completely relieved of the sharp, shooting pain radiating from the tonsillar fossa to the ear. The left half of the soft palate and the left half of the upper pharyngeal muscle were paralyzed. The taste senses on the left half of the tongue were diminished. The patient had no difficulty in speaking or in drinking liquids, but had slight difficulty in swallowing a large formed bolus of food. He could yawn and drink cold water without precipitating the attacks. He had, however, an aching pain in the left eye, radiating horizontally backward above the ear, and this was associated occasionally with lacrimation and a stuffiness in the left nostril, giving him the impression that he was "catching cold." The subsequent postoperative course has been satisfactory so far as the shooting pains are concerned, but the patient complains a great deal of distress in the eye.

INTRACRANIAL DIVISION OF THE GLOSSOPHARYNGEAL NERVE

One patient I had operated on for glossopharyngeal neuralgia complained of recurrence of pain, which influenced me to develop an intracranial approach for division of the glossopharyngeal nerve. The technic worked out on a cadaver can, I believe, be employed for the relief of the disease with accuracy and safety.

The incision is that used in performing unilateral cerebellar decompression; it is carried from the spine of the atlas upward to the external occipital protuberance; then, in the form of a horseshoe, it is carried up to the lateral sinus and down over the mastoid to its tip; the skin and muscles are reflected in the same manner as they are reflected for cerebellar decompression, and the bone is removed upward and laterally, exposing the lateral and sigmoid sinuses. The bone is removed mesially and downward until the external occipital crest, the posterior condyloid foramen and the margin of the foramen magnum are approached; the dura is then incised and reflected mesially with a basal flap along the

crest of the occipital bone (Figs. 4 and 5). If the cerebellar lobe does not displace easily, one can either drain the posterior cistern or the posterior horn of the lateral ventricle through a separate incision above the lateral sinus. Before elevating or displacing the cerebellar lobe, it is well to cover the cortex with strips of cotton; then with the illuminated retractor elevate the lateral lobe of the cerebellum, when the seventh and eighth cranial nerves can readily be seen entering the internal auditory meatus (Fig. 6). Inferior to these nerves and slightly more superficial, one will observe the ninth, tenth and eleventh nerves entering the jugular foramen; on closer observation, the ninth and tenth nerves are found to be short and to pass at almost right angles from the medulla, while the spinal accessory is longer and enters the foramen

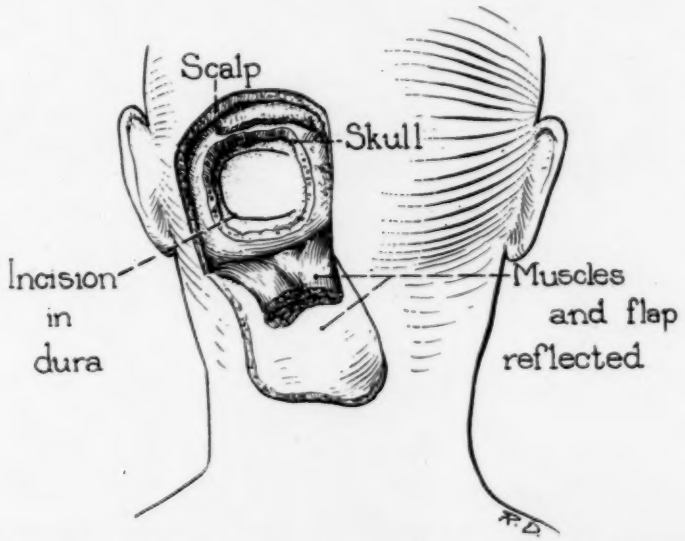

Fig. 4.—Incisions in scalp and dura for cerebellar approach of glossopharyngeal nerve.

in an oblique course. Further, it will be seen that the glossopharyngeal enters the foramen at the upper portion, and is separated from the vagus by a small dural band which is less than 1 mm. in width, but definite enough to permit one to pass a small right angle ganglion knife between the fibers of the vagus and glossopharyngeal nerves, thus facilitating a sharp section of the glossopharyngeal nerve (Fig. 7). Experience in the surgical treatment of acusticus tumors enables the surgeon to find that the intracranial division of the glossopharyngeal nerve is less difficult, and that the field is more easily exposed. Closure of the wound is the same as that employed in cerebellar explorations. The operation could be performed under combined anesthesia with a

small quantity of ether given just before the division of the glossopharyngeal nerve is effected. In spite of the fact that my experience in peripheral avulsion for glossopharyngeal neuralgia is very small, I would suggest the intracranial division of the glossopharyngeal nerve in preference to the extracranial division, on account of the necessity of

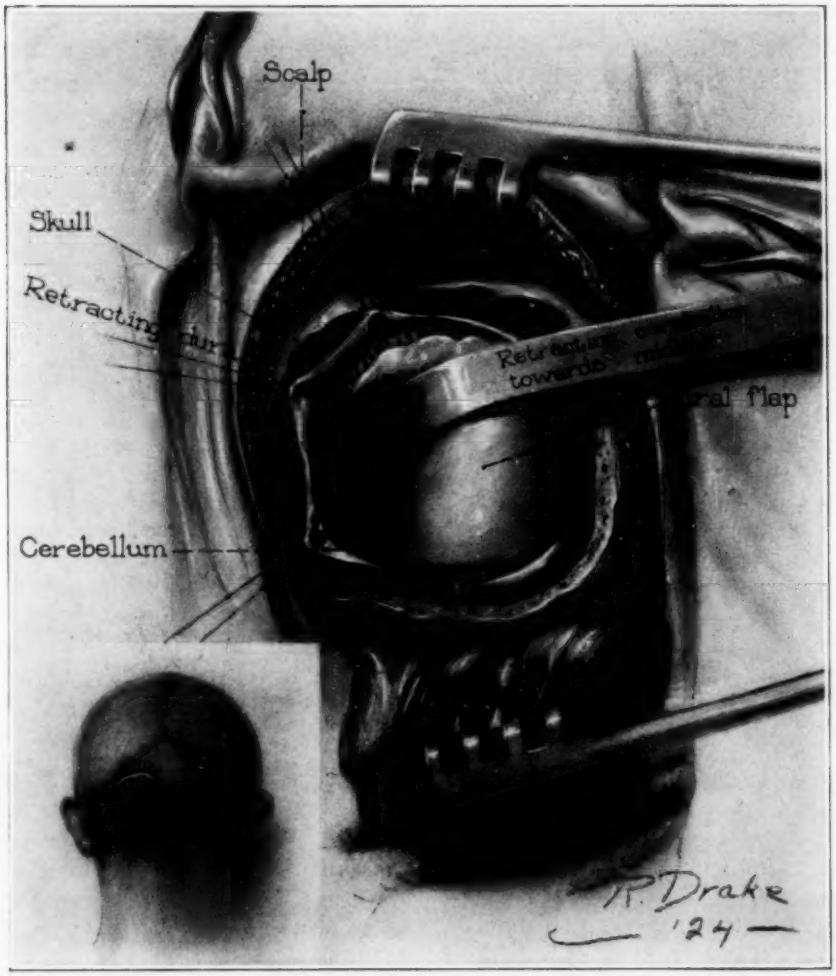

Fig. 5.—Cerebellar approach for the intracranial division of the glossopharyngeal nerve for glossopharyngeal neuralgia.

the major operative procedures, and the possibility of recurrence following the extracranial operation.

Glossopharyngeal neuralgia is a disease with a definite entity, similar to trifacial neuralgia in its course, and probably having its origin in the superior ganglion of the glossopharyngeal nerve; it can be relieved

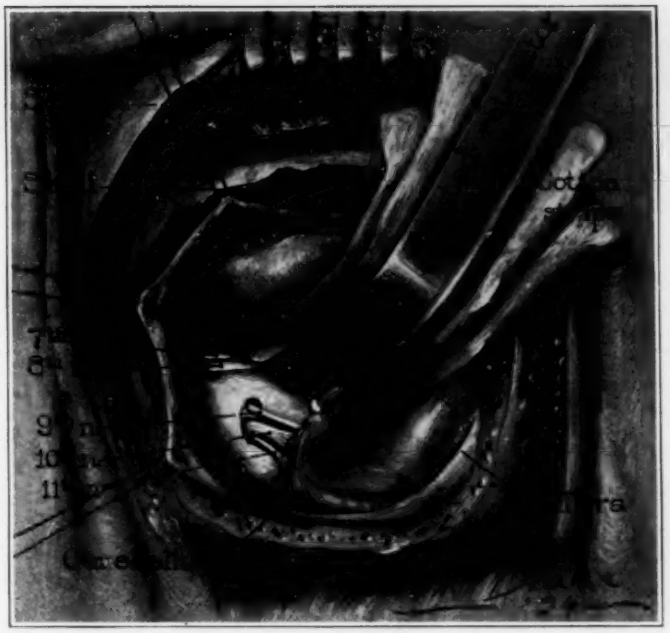

Fig. 6.—Exposure of the seventh, eighth, ninth, tenth and eleventh cranial nerves at the point of exit from the skull, preliminary to the division of the glossopharyngeal nerve.

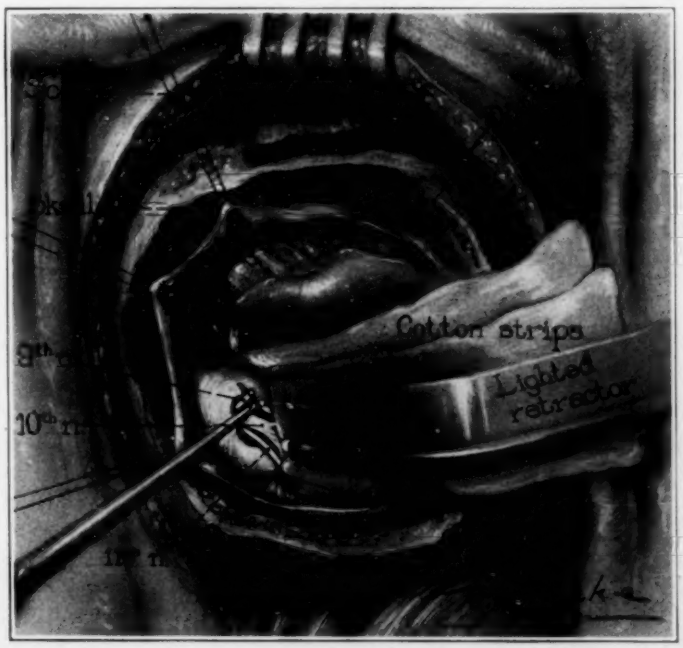

Fig. 7.—Division of the glossopharyngeal nerve through the intracranial approach.

temporarily, and possibly permanently, by an extracranial avulsion of the glossopharyngeal nerve and part of the ganglion, but division of the root intracranially should afford a permanent cure, which procedure I would advise since the technic is no more formidable than an extracranial avulsion.

CARDIOVASCULAR PHYSIOLOGY OF THE MENTAL DEFECTIVE *

G. S. STEVENSON, M.D.

MINNEAPOLIS

The conception of the feebleminded person as a mental child and a physical adult serves a definite and practical purpose, but cannot be taken over as a biologic concept. The psychobiologic differences between mental 6 year old and chronologic 6 year old are so great, even if they have many similarities, that protest after protest follows the attempt to drag the mental age theory into every process having to do with the feebleminded. The understanding of the meaning of feeblemindedness and of its biologic relationship will never come through perfection of one artificially detached line, but may be approached by recognizing degree in many diverse and interdependent qualities. More concretely, the greater the number of qualities we can consider in a person, the closer will our results approach those of practical experience. This must be so, since the individual is indivisible in his behavior. And so when we consider examinations of intelligence, of emotional stability, of psychophysical response, of vegetative balance, of the physical status and of anthropologic make-up and line them up with sex, age, race, etc., we are approaching closer to accuracy than when we consider any one of these. Because, generally speaking, with the feebleminded the intelligence weighs heavier than does the physical status of the individual, intelligence testing has attained a high ranking in this field. But, for this same reason whoever approaches the problem from many angles will be similarly successful. There has been a failure, to date, adequately to approach the question—What are the physiological peculiarities of the mental defective? It has long been recognized that developmental anomalies occur in unusually large numbers among the feebleminded, and a few well founded physiologic anomalies or inferiorities have been demonstrated (vital capacity, muscle strength), but on the conditions of grosser anatomy and the more highly integrated physiology, the psychology of the individual, comparatively little has been done.

One pitfall in the investigation of the feebleminded has been the failure to recognize the possibilities of classification or the significance of this from the standpoint of disease entity and etiology. Certain types—cretin, mongolian, cerebral traumatic or hemorrhagic, and

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hydrocephalic—have been recognized as distinct disease entities with, in some cases, known etiology. But these have at times been grouped with the less well defined types in drawing conclusions in which disease type as well as mental level should have been considered. We read of carefully performed postmortem work, for instance, on glandular conditions in which results have been taken as a whole without regard for type of feeblemindedness and without regard for cause of death. This is less a criticism of methods than a statement of the status of our understanding of feeblemindedness. For, should we limit attention to the type known as hereditary, we again find ourselves in the position of knowing even less certainly whether we face a unity or not, feeling strongly that we do not, but at the same time unable to separate the varieties. Not only are we concerned about the unity of the thing studied, but also about the purity of it. For instance, the arched palate is probably an insignificant concomitant or impurity, or a part of certain cases, but when the coincident anomaly is one of emotional balance, we have a deviation from the normal which may occur in all degrees; and depending on what we are studying, we have a variable which may or may not seriously affect results.

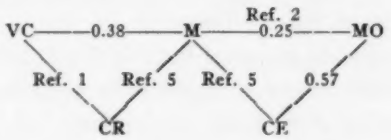
When it comes to drawing conclusions, the control of cases has always been the "monkey wrench in the works." These feebleminded studied in institutions may make our normal persons look like poor specimens on absolute comparison. But many feebleminded children have been put into exceptionally fine condition for them, because of the regularity of institution life. A method of control that has been made good use of is that of comparing higher with lower grades of feebleminded. In doing this we have to assume that because the feebleminded as a class pass imperceptibly into normality in their social adaptations, they do also in their other characteristics, and we make use of the reasoning that the low is to the high grade feebleminded as the high grade feebleminded is to the normal. We have to assume that we study quantitative rather than qualitative differences and that all the characteristics are relative.

The study of the feebleminded from the biologic standpoint has been hampered by the injection of an arbitrary social criterion, which has given to this group a qualitative rather than its deserved quantitative differentiation from the normal. Considerable effort has been made to define and criticize definitions of feeblemindedness, yet every definition has its objections. For scientific purposes, why attempt to define something that has no set boundaries, one that actually is not delimited, and has an obscure borderland region? In this case, it is preferable to stake a central point which will indicate the field rather than a wall which will delimit it. Suppose we should select one quality, such as feeblesightedness, and define it as that degree which will prevent the

individual from accomplishing a certain job. We should have a useful classification as far as that job goes, but biologically we should have blocked ourselves if we carried this over as a working hypothesis. We should want rather to examine all grades from superior vision to total blindness. This quantitative difference is a fact within feeblemindedness and within normality, and we assume it to be a fact between feeblemindedness and normality.

CIRCULATION

For the study of circulation in the feebleminded, certain investigations formed a background.



Studies made on the feebleminded show a correlation of about 0.38 between defective mentality (*M*) and defects in vital capacity (*VC*). Vital capacity is further closely correlated with cardiac strength (*CR*) in pathologic cases.[1] Dynamometer results (*MO*) have been found to correlate around 0.25 with mental age, and Martin has found this same relation to exist among normal persons graded on their industrial level.[2] He used a more intricate test of muscle strength.[3] This problem is closely connected with a greater endurance in persons of higher mentality. Exercise tests (*MO*) and circulatory efficiency tests (*CE*) have also been found to have a significant correlation.[4] Since mental level and circulatory reactions both show a definite relation to muscle output, it would be interesting to know relations between circulation and mental level. Since with a maximum effort there is a variable muscle response, depending on the grade of feeblemindedness, what would be the result, measured by circulatory response, of the same job, that is, approximately the same muscle response, allowing the effort to be variable? Since, as has been said, it is unpractical to get normal controls for observations of the feebleminded where training and the mode of life are important factors, the method of comparing high and low groups of feebleminded was resorted to.

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1. Wilson, May G.: Exercise Tolerance of Children with Heart Disease, J. A. M. A. **76**:1629 (June 11) 1921.

2. Martin, E. G.: Personal communication.

3. Martin, E. G.: Tests of Muscular Efficiency, Physiol. Rev. **1**:454 (July) 1921.

4. Finkelstein, W., and Williams, J. F.: Correlations of Efficiency Tests, J. A. M. A. **78**:1454 (May 13) 1922.

METHODS

For the circulatory efficiency test, as conducted by Schneider [5] (Table 1), two groups were formed—one consisted of all the men available between 20 and 25 years of age and the other of those between 25 and 35 years of age. Those having physical handicaps, such as paralysis and cardiac disorders, and those included in the more well defined groups, as mongolians, were eliminated; those having colds were not used until well. Those under 20 years of age were considered too unstable to be good working material.[6] Subsequent trial on a small group confirmed this. Those over 35 were few and by stopping at 35, the influences of arterial changes were largely avoided. It would have been desirable to have eliminated as many variable factors as possible, but the available material limited the possibilities of selection.

A further desirable selection might have been made on the basis of adjustment to existing conditions, adaptation, had it been possible to make some quantitative rating of this character and to have some basis for determining how much to allow to be present. But our ability to distinguish between an acquired and a congenital or constitutional psychopathic tendency, and our ability to rate the rôle of psychopathy as a part of feeblemindedness is now so limited as to present a practical obstruction to the handling of it in this regard.

Schneider's test was chosen less because of the collective rating than because of the fact that it is based on important circulatory observations. The observations individually were given more attention than the collective rating. Three tests were made on each person, and if these varied more than four points in the final rating, the subject either was rejected or the tests were repeated until three consecutive ratings were within four points of each other. Often the first rating was low when the boy was timid or anxious, but in this case the fourth test was usually satisfactory. As a check, the two groups of boys were examined separately.

RESULTS OF CIRCULATORY EFFICIENCY TESTS

An analysis of circulatory data brings out some interesting comparisons. In Table 1, the figures presented are, unless otherwise stated, averages of all of the thirty-seven and thirty-six cases in the low and high groups, respectively.

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5. Schneider, E. C.: A Cardiovascular Rating as a Measure of Physical Fatigut and Efficiency, J. A. M. A. **74**:1507 (May 29) 1920; Further Observations on a Cardiovascular Physical Fitness Test, Mil. Surg. **52**:18 (Jan.) 1923.

6. Williams, J. F.: Correlations of Efficiency Tests, J. A. M. A. **80**:16 (Jan. 6) 1923.

The essential points are: 1. The average pulse rates on lying, standing and exercise of the low group are higher than those of the high group (*A, C, E*). Tabulating the number of instances for each recorded pulse rate shows the mode of the high grade group to lie at 66 (eighteen cases) and that of the low grade group to lie at 72 (fourteen cases). The grouping of the pulses lying down, as shown in Table 1, *B*, brings this out. The variability is greater in the low grade group ranging from 42 to 96 than in the high grade group ranging from 48 to 87. This variability holds also for the standing and exercise pulses, but the standing pulse likewise shows the same lower mode found with the lying down pulse for the high group. 2. The tendency to pulse change on change of position is greater in the low grade group

TABLE 1.—*Results in Circulatory Tests*

	Low	Readings	High	Readings
A. Lying pulse	71.8	111	65.0	106
B. Standing change for lying pulse of				
Under 51	13	6	12	3
51 to 63	16.6	27	15.2	49
66 to 78	12	56	10.4	45
81 to 90	8.5	18	−2	7
Over 90	6.8	5	0	0
All	11.8	...	11.2	...
C. Standing pulse	83.6	...	76.2	...
D. Exercise change for standing pulse of				
Under 60	14	3	8.5	2
60 to 69	14	12	16.9	30
70 to 79	12	26	12	32
80 to 89	12	35	9.2	27
90 to 99	10.9	30	4.9	15
100 to 109	4.7	3	20	1
Over 109	0	2	0	0
All	11.7	...	11.9	...
E. Exercise pulse	95.5	...	88.0	...
F. Lying systolic blood pressure	119.5	...	114.1	...
G. Change on standing	−4.7	...	−4.5	...
H. Standing systolic blood pressure	114.8	...	109.6	...
J. Average score	11.0	...	12.1	...

(Table 1, *B*). This is more than appears in the average figures for the whole groups as is shown by an analysis of the changes according to the different lying down pulse rates. The rule of pulse change is that the higher the initial pulse, the less the change; hence the excess of cases in the low grade group with a higher initial rate, and consequently a smaller change makes the difference less between the average changes than actually exists. This is shown in Table 1, *B*, in which the two groups are compared rate for rate. Expressed in terms of correlation between lying pulse and pulse rise on standing, the figures are: —0.44 and —0.57 for high and low grade groups, respectively. Changes from standing pulse to exercise pulse, while having somewhat the same relations as those existing between lying pulse and standing pulse, have certain differences also. On the whole, there is probably a tendency toward more rise, considering the height of the standing pulse as in the foregoing, in the low grade group. The correlation between lying pulse

and pulse on exercise shows a rather marked change from the one previously stated. For the high grade group, it had risen to —0.55, while for the low grade group the correlation practically disappeared, falling to —0.07. It is worth noting how few cases occurred in parts *B* and *D* of Table 1 in the higher pulse rates with the high grade groups. This is what would be expected with a lower mode. 3. The average systolic blood pressure of the low group was higher than that of the high grade group, both on lying and on standing. But the fact which is important as an indicator of the status of the efficiency of the circulatory system, the change in blood pressure on assuming standing posture, was practically the same in both, and was a fall. The slight difference is, however, significant, since a lower blood pressure usually tends to rise less than the higher on change of posture.[7] 4. Cases rated according to the Schneider score gave a lower figure for the low than for the high grade group. 5. It was stated previously that in some cases the test had to be repeated in order to get scores agreeing closely enough to be acceptable. The repetitions required for this were three times as frequent among low grade boys as among high grade boys.

Briefly summarized the results of the circulatory observations are:

1. There is a lower recumbent, standing and exercise pulse rate average in the high grade group.
2. There is a greater pulse response in the low group to a change of posture and possibly also to exercise, but the pulse of the low grade group by exercise loses all correlation with the preceding pulse.
3. The low group has a higher systolic blood pressure.
4. Both groups show a systolic blood pressure fall on rising from a reclining posture.
5. The test rating is lower in the low grade group.
6. The lower grade group shows a greater pulse distribution and a necessity for more repetitions of the tests.

CARDIAC RESERVE TEST

In order to understand further the blood pressure responses to exercise, the "cardiac reserve test" as used by Barringer[8] and Wilson was employed. This test consists of the following steps: (1) initial systolic blood pressure reading with patient at rest; (2) standard dumb-bell exercise; (3) immediate rapidly recorded successive readings of blood pressure for two minutes and again at three minutes.

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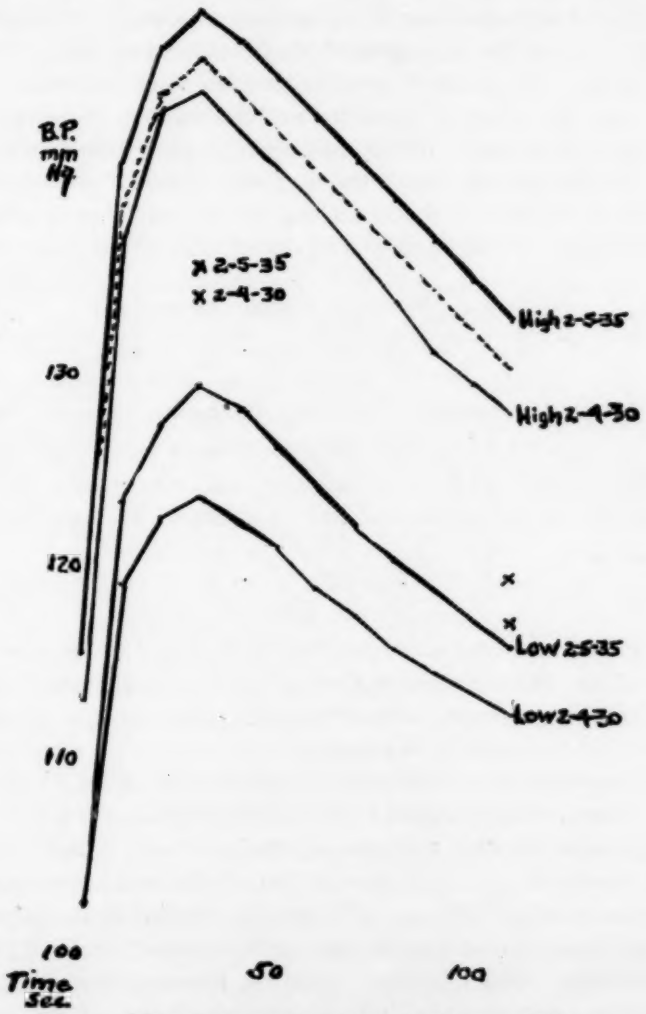
7. Schneider, E. C., and Truesdell, D.: A Statistical Study of the Pulse Rate and Arterial Blood Pressures in Recumbency, Standing and After a Standard Exercise, Am. J Physiol. **61**: 429 (Aug.) 1922.

8. Barringer, T. B.: Studies in the Heart's Functional Capacity, Arch. Int. Med. **20**:829 (Dec.) 1917.

The customary response is an ascent of blood pressure for a period up to thirty-five seconds after the cessation of exercise, followed by a descent which is completed in two minutes. As the cardiac reserve is overtaxed by increasing the exercise, the rise of blood pressure becomes more prolonged, and it descends more slowly.



Curves of blood pressure response in 15 year old boys.

Two groups were employed for this test. Group 1 consisted of boys 11, 12 and 13 years of age, but middle normal in height and weight. Five of these had an intelligence quotient below 0.5, and four had an intelligence quotient above 0.8. In the first series of exercises, two 4 pound dumb-bells were swung twenty times, designated as 2 by

4 by 20. In the second series, the exercise was 2 by 4 by 30. Group 2 was composed of boys about 15 years of age and of middle normal height and weight. Five had an intelligence quotient below 0.45 and five above 0.75. Their exercises were 2 by 4 by 30 and 2 by 5 by 35. Each exercise was repeated ten times by each boy on five different days.

The data thus obtained were treated in two ways. By interpolation, an average of the ten trials at ten second intervals was obtained, and from this a curve for each series for each child was made. Composite curves of the high and low grade boys were made separately for each group. Thus the effect of the added exercise could be shown graphically more or less accurately. While this curve drawn from averages based on time gives a general idea of the reactions as shown in the illustration, it is made inaccurate by the smoothing of the peak due to small variations in the time of the peak. The dotted line in the chart represents

TABLE 2.—*Results of Cardiac Reserve Test*

	Exercise	A Time of Peak, Seconds	B Rise of Peak, Mm. Mercury	C Time of Plateau, Seconds	D 2 Minute Failure, Mm. Mercury
12 year low.....	2-4-20	36.0	13.7	29.5	6.2
	2-4-30	36.2	19.6	17.1	10.1
12 year high.....	2-4-20	27.6	18.6	30.0	7.2
	2-4-30	28.4	23.0	25.1	12.6
15 year low.....	2-4-30	27.7	22.2	10.5	9.7
	2-5-35	26.9	27.2	13.3	12.5
15 year high.....	2-4-30	21.8	32.7	13.6	13.4
	2-5-35	21.9	33.7	19.7	15.3

the curve dropped to the same initial point as 2 by 4 by 30 to make comparison easier. The crosses represent the curve peaks and falls of the high grade cases dropped for comparison with the low grade to the initial level of the latter (103 mm.).

Data representing actual average heights and times of peaks and other prominent features give a more accurate idea of the reaction and the comparisons between the groups. It was necessary to adopt certain rules in determining the two points: end of rise and beginning of fall. These rules were as follows: The peak is the maximum point if the rise is continuous, otherwise the first point within 2 mm. of the maximum is chosen. The fall begins with the last maximum reading if it is continuous, otherwise the last point within 2 mm. of the maximum is chosen as the beginning of the fall.

Results of the Test.—Inspection of the data of Table 2 and general checking up with the chart reveals that: (1) The blood pressure in the low grade group rose less. Compare the couplets in *B* for each group of boys and each exercise. (2) The low grade groups responded more to an increase of work. Compare the differences in the couplets

of *B* for each group of boys. (3) The blood pressure in the low grade group rose for a longer period than the high grade. Compare the couplets in *A* for each group of boys. (4) The plateau was shorter in the low grade group. Compare the couplets in *C* for each group of boys. (5) The blood pressure returned closer to the initial with the low grade group in two minutes, but the percentage of return was higher in the high grade group. Compare couplets in *D* for each group of boys.

Certain similarities are worth mentioning: (1) The blood pressure rose with more work. Compare the parts of each couplet in *B*. (2) The period of rise tended to lengthen with more work. Compare the parts of each couplet in *A*. (3) The blood pressure return was slower with more work. Compare the parts of each couplet in *D*.

VEGETATIVE SENSITIVITY TESTS

Ultimately any consideration of cardiovascular responses should include also some attention to the control underlying these responses. While we know little of the vegetative nervous system itself, we are able to recognize certain manifestations of its activity and of its sensitivity, and these we can study. When we speak and think of this system, we really refer to these activities and not to the neurons behind them. With this in view the reactions to the three drugs—suprarenal extract, atropin, and pilocarpin—and the oculocardiac reflex were recorded.

In the case of the drug tests, a twenty-five minute control period was employed, during which pulse, respiration, systolic and diastolic blood pressure were recorded at five minute intervals. At the end of twenty-five minutes, the drug injection was made, the amount being calculated by body weight and amounting to: suprarenal extract (1:1,000) 0.5 c.c.; atropin, 1/150 grain and pilocarpin 1/12 grain for a person of 55 kg. body weight. The readings were then continued at two and one-half minute intervals for fifteen minutes, and then at five minute intervals to the end of one hour after injection. Other drug effects were looked for.

The three drug tests were made in each of seventeen cases of uncomplicated feeblemindedness in boys, mostly middle and high grade imbeciles, all corresponding with the normal middle percentages in height and weight and being from 16 to 18 years of age. A few tests were made in other cases in which these requirements were not made.

In the case of the oculocardiac reflex, pressure of varying degrees (measured) was made, and the pulse change was recorded for each. The maximum change was obtained in most cases with a pressure between 175 and 225 gm. to the square inch. Ten observations were made on the regular group and four on the unselected cases.

The results obtained from the drug and Aschner tests are reported together in Table 3. The outstanding features are: (1) There is an evident sensitivity to suprarenal extract, with much less response to atropin and pilocarpin. (2) The oculocardiac reflex is predominantly one of pulse increase relative to the normal response. Both of these reactions point to a sympathetic sensitivity, which it may be concluded is here present. In connection with these tests, it will be interesting to note the results to be discussed later, of the administration of atropin to patients suffering from enuresis.

TABLE 3.—*Results of Vegetative Sensitivity Tests*

	Cases	Response
Suprarenal extract, 21 cases.....	5	Change less than 20 in pulse or pulse pressure
	4	Change 20 to 24 in pulse or pulse pressure
	2	Change 25 to 29 in pulse or pulse pressure
	1	Change 30 to 35 in pulse or pulse pressure
	7	Change over 35 in pulse or pulse pressure
Atropin, 18 cases.....	13	No significant response to drug
	3	Moderate response to drug
	2	More marked response to drug
Pilocarpin, 18 cases.....	8	No significant response to drug
	7	Moderate response to drug
	3	More marked response to drug
Aschner, 13 cases.....	2	Slowing of over 12 points (16, 20)
	4	Slowing of 4 to 12 points (4, 7, 9, 12)
	7	Slowing of under 4 points (3, 3, 1, −3, −3, −6, −9)

INTERPRETATION OF RESULTS

To draw general conclusions from the preceding findings concerning the circulatory functions of the feebleminded would be hazardous. There are certain indications that the feebleminded of the low grade have reacted like untrained persons when compared with those of higher grade. There is no difference in the daily work of the two groups to account for this. The lower pulse, less pronounced pulse response, lower blood pressure, higher test score and fewer repetitions are all points in line with this idea. Furthermore, the feebleminded person probably spends himself sooner. The falling off in correlation between original pulse and exercise pulse in the low grade group may mean that previous responses have raised the pulse so near its maximum that the pulse which responds to exercise has no leeway to assume its normal relations to the preexisting pulse, hence loses correlation with it. The prolongation of the rise of blood pressure after exercise in the low grade cardiac reserve group and the greater early response in the high grade group shows a relatively closer approach to the limit in the low grade group. It would have been desirable to have in this connection a series of graded exercises both easier and harder than those used. The greater fall in the standing blood pressure of the low grade group is indicative of relative inefficiency. Normally, a pressure of 118 would

fall less (or rise more) than one of 114. The fact that both fell is significant of poor vasomotor compensation. Schneider[9] found no greater failure in scoring in this than in other parts of the test. We may seriously consider whether the term "no guts" may not in part mean a lack of splanchnic tone to keep the pressure even and the blood well distributed. While the general test scores are good, in spite of the blood pressure failures, it probably indicates that the regularity of institution life has kept the boys in good form and possibly close to the maximum. Had we normal controls under identical conditions, it is probable that they would show even better results.

Certainly the drug tests are indicative of abnormal sensitivity of the thoracolumbar portion of the vegetative nervous system, and the Aschner results are quite in line with this. It is interesting in this connection that in another experiment in which sixty patients with enuresis were given atropin, the condition was improved at least 25 per cent. in 30 per cent. of the cases. There were a number of cases in the groups which it was felt were misplaced. One boy, particularly, who had a pulse rate of 42, 48, 56 on lying, standing and after exercise, respectively, was in the low grade group. But industrially this boy rated high. It is a question whether mental age alone gives a close biologic classification here. Had it been possible also to grade and to rate the emotional balance and industrial efficiency, an even more satisfactory comparison between the groups would have resulted. The psychophysical inferiorities of the feebleminded are probably not merely dependent on a defective or insufficient cerebral impulse, but there is as well a paralleling inferiority in the integrations of the vegetative nervous system. To this we may attribute the instability of circulatory response of the feebleminded.

9. Personal communication.

RESULTS OF BASAL METABOLISM TESTS IN ONE HUNDRED MENTAL CASES*

CLIFFORD B. FARR, M.D.

PHILADELPHIA

Boothby, Sandiford [1] and McCann [2] have, since the beginning of the present year, reviewed the literature of basal metabolism and calorimetry in exhaustive fashion and have envisaged the clinical aspects from every angle. The general topic therefore does not call for extensive comment at the present moment; hence I am presenting observations made between Nov. 6, 1922, and Dec. 28, 1923, in mental cases, in the form of charts, with the briefest explanations possible and such tentative conclusions as seem justified. The charts exhibit basal metabolism findings, as percentages of the normal, in 100 cases, arranged in order of heat consumption from the lowest to the highest. These cases were selected from a total of 134, not for clinical reasons, but because of the reliability of the instrumental findings. In each instance, observations were made on several different occasions, and the lowest figures obtained, if not otherwise open to criticism, were chosen as probably basal. Care was taken to observe all the usual precautions as to preliminary fasting and rest. On the whole, the patients showed a degree of cooperation that compared well with that found in the general run of hospital cases. Naturally, we did not try to determine the oxygen consumption in maniacal and agitated patients.

We used the Benedict appartus as modified by Roth, and found it both simple and, as far as we could determine, accurate. Soon after beginning the tests, we verified the accuracy of the calibration. The "flutter" valves, which in this machine permit the motor to be eliminated, gave us no trouble by leakage. The only novelty about our apparatus was a suspension of the mouthpiece by adjustable cords from an overhead rod, thus doing away with any rigid connection between the patient and gasometer and protecting the machine from jarring or injury. On every test day, we recorded the results of two or more periods, each consisting of three or more overlapping, six-minute

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* Read by title before the Philadelphia Neurological Society, March 28, 1924.

* From the Pennsylvania Hospital, Department for Mental and Nervous Diseases.

1. Boothby, Walter M., and Sandiford, Irene: Basal Metabolism, Physiological Rev. **4**:69, 1924.

2. McCann, William S.: Calorimetry in Medicine, Medicine **3**:1, 1924.

intervals. Some patients did best on a first test, afterward becoming irritable; others seemed to profit by experience and were more composed on a second trial.

Chart 1 shows (solid dot line) 100 cases, arranged, at equal intervals from left to right, according to the height of the metabolism. Limits of the normal, 10 per cent. plus or minus, are shown by heavy lines. Twenty-eight per cent. of the cases were below normal and 13

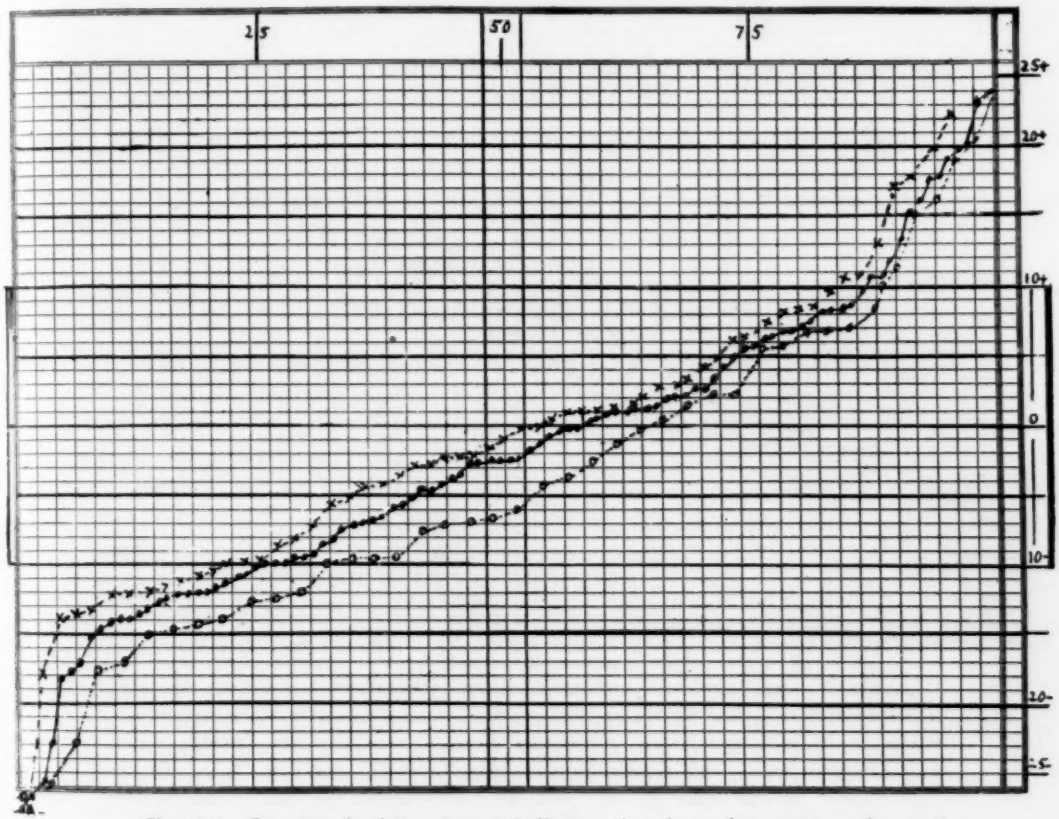

Chart 1.—One hundred basal metabolism estimations, forty-two males and fifty-eight females. Findings below the lower normal limit in women were 22 + per cent.; in men, 31 per cent.; findings above the normal limit in women and men, 12 per cent. The X line is the curve for women; the solid dot line, that for the total number of cases, and the hollow dot line, that for men.

per cent. above, a total of 41 per cent. showing a deviation from the normal in one direction or the other. The forty-two male patients (hollow dot line) are arranged at equal intervals parallel to the foregoing and are seen to average somewhat lower; the fifty-eight females (x line), on the other hand, are slightly above the combined curve.

Chart 2 shows the same series of cases (solid dots) with separate curves according to diagnosis. The upper line, indicated by hollow squares, represents twenty-seven patients (twelve males and fifteen

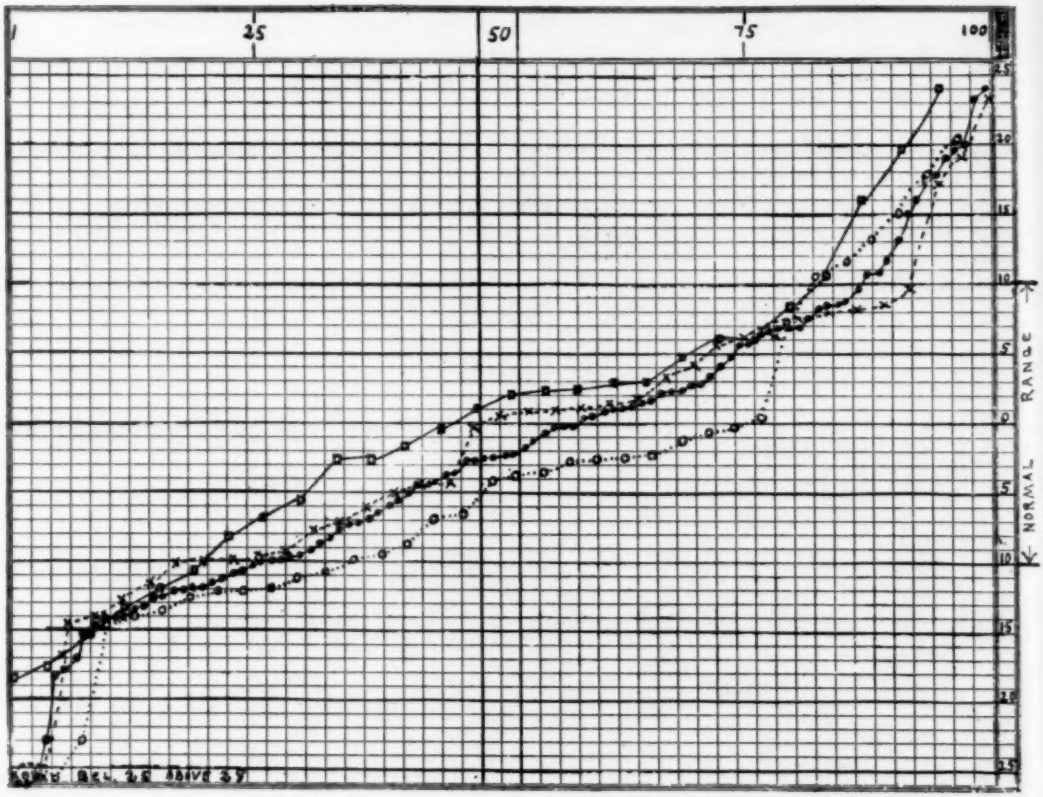

Chart 2.—One hundred basal metabolism estimations, the curves showing the distribution of findings in various types of mental disorder. The upper line, made up of hollow squares, represents twenty-seven patients (twelve males and fifteen females) with miscellaneous psychoses, 22 per cent. being 10 per cent. below normal and 15 per cent. as much above normal. The X line represents thirty-eight patients (thirteen males and twenty-five females) with mood disorders, 24 per cent. being below normal and 8 per cent. above. The solid dot line represents the total number of cases (one hundred—forty-two males and fifty-eight females), 28 per cent. being below normal and 13 per cent. above. The hollow dot line represents thirty-five patients with dementia praecox (seventeen males and eighteen females), 37 per cent. being below normal and 17 per cent. above. Of five patients who presented a metabolism of —20 or lower, two depressed patients (one man and one woman) were profoundly undernourished, three dementia praecox patients (all men) were spare rather than emaciated. Of nine cases (three in each category) with a basal metabolism of 15 + or more, one manic patient manifested other evidences of hyperthyroidism, one dementia praecox patient had an enlarged thyroid and a second had a slight tremor.

females) with miscellaneous psychoses, 22 per cent. of which were 10 per cent. minus or lower, and 15 per cent., 10 per cent. plus or higher. This curve is slightly above the combined curve. The curve indicated by x's represents thirty-eight patients (thirteen males and twenty-five females) with mood disorders (involutional melancholia and manic-depressive insanity) corresponding closely to the curve for the whole group. The lowest line represents thirty-five patients (seventeen males and eighteen females) with dementia praecox. Most of these cases were of relatively short duration, with little deterioration. In this group, 37 per cent. were 10 per cent. minus or lower, and 17 per cent. were 10 per cent. plus or higher; in other words, more than 50 per cent. abnormal as compared with 30 per cent. in the group of mood disorders. If cases with definite thyroid disease are excluded, the figures are not appreciably changed.

These findings suggest some endocrine imbalance in dementia praecox, but our not very extensive therapeutic trials have shown no definite improvement after thyroid extract and thyroxin. The investigations in this field cited by McCann,[2] and, in a recent article, by Bowman and Grabfield[3] showed a similar diminution of metabolism in mental cases and particularly in dementia praecox. With the exception of Lewis and Davis, none of them has had a satisfactory experience with thyroid treatment. Inanition and physical inactivity have not been shown to be important factors. While basal metabolism estimations have so far not proved very informing, it seems to me that they should be continued, preferably in combination with other vital function studies, as a guide to experimental endocrine therapy.[4]

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3. Bowman, K. M., and Grabfield, G. P.: Basal Metabolism in Mental Disease, Arch. Neurol. & Psychiat. **9**:358, 1923.

4. Rowe, Allan Winter: Vital Function Studies, Boston M. & S. J. **190**: 291, 1924.

INSULIN AND MENTAL DEPRESSION *

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JOHN P. PARSONS, M.D.
AND
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In our work with diabetic children, we have always been impressed with the marked symptoms of depression shown by these patients. We have observed that the most noticeable reaction from the use of insulin was the clearing of the depression. This observation led us to make some investigations into the possible effect of insulin in states of true mental depression. Raphael and Parsons have shown that there is a disturbance of the normal utilization of glucose in manic-depressive psychosis. In the depressive phase, there is a delayed utilization of glucose comparable to that obtained in diabetes mellitus, while in the agitational phase the glucose utilization curve is flat or has a tendency to fall below that of a normal person. This would seem to indicate an increased utilization of glucose. The following investigations show the effect of insulin on the glucose absorption and utilization in the depressive phase of manic-depressive psychosis.

METHODS

All observations were begun on patients who had fasted since the previous evening meal. Samples of blood were taken at intervals indicated in the tables and on the charts. The Folin and Wu blood sugar method was employed. The glucose was given by mouth. The amount given was determined by the weight of the patient in kilograms multiplied by 1.75. The amount of water was calculated by multiplying the amount of glucose by 2.5.

EXPLANATION OF CHARTS

The blood sugar percentage scale appears to the left of the chart. The first space following records the blood sugar control observations. The hours after the glucose was given are represented by the spaces that follow, 1-2-3-4-5-6. These spaces are divided into four divisions which represent quarter hours. The curves are numbered, Number 1 always being the control glucose curve without insulin. The succeeding curves 2-3-4 are those obtained with insulin. The dots on the curve represent the percentage of sugar in the blood at the time recorded on the chart. The curves are made to pass through these points as closely as possible and represent the approximate course of the absorption of sugar into the blood and its utilization.

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REPORT OF CASES

CASE 1 (Chart 3).—Bertha W., a white woman, aged 42, single, was admitted to the State Psychopathic Hospital, Aug. 16, 1922, with a diagnosis of manic-depressive psychosis, depressed phase, first attack. She weighed 56 kg. The father was reported to have been alcoholic, and one sister and paternal grand-uncle to have suffered "nervous breakdowns." One cousin was said to have been insane—exact condition unascertained. The previous personal history seems to have been essentially negative, except that the patient appeared always to have been of a somewhat sensitive reticent personality type and much inclined to worry and brood.

The present trouble began in January, 1922, with depressional features, and on examination at the State Psychopathic Hospital she was found to be suffering from a depression of the typical manic-depressive type, evidencing marked affective depression, thought difficulty, psychomotor retardation and delusions of a depressional type; thus ideas of unworthiness, personal sin and impending misfortune were manifest. Somatically, aside from a certain undernutrition, no deviation was found. The routine laboratory examinations were uniformly negative. The patient's course continued as described in the foregoing without improvement until her discharge from the hospital, Oct. 18, 1923.

TABLE 1.—*Glucose Absorption Utilization Observations in Case 1*

Date	Time		Blood Sugar Percentage
		Without Insulin	
7/12/23*	8:10 a.m.	Fasting blood control	0.10
	8:15 a.m.	99 gm. glucose by mouth, 248 c.c. water	
	9:30 a.m.	Blood sample	0.15
	10:30 a.m.	Blood sample	0.22
	11:30 a.m.	Blood sample	0.171
	1:00 p.m.	Blood sample	0.150
		With Insulin	
7/17/23†	7:50 a.m.	Fasting blood control	0.10
	7:55 a.m.	2 units insulin, subcutaneously H10	
	8:25 a.m.	Blood sample	0.09
	8:26 a.m.	99 gm. glucose by mouth, 248 c.c. water	
	9:25 a.m.	Blood sample	0.20
	10:25 a.m.	Blood sample	0.15
	11:25 a.m.	Blood sample	0.08
	12:25 p.m.	Blood sample	0.09
	1:25 p.m.	Blood sample	0.07
7/20/23‡	8:00 a.m.	Fasting blood control	0.09
	8:35 a.m.	Fasting blood control	0.10
	9:00 a.m.	2 units insulin, H10, subcutaneously 99 gm. glucose by mouth, 248 c.c. water	
	10:00 a.m.	Blood sample	0.184
	11:00 a.m.	Blood sample	0.166
	12:00 a.m.	Blood sample	0.108
	1:15 p.m.	Blood sample	0.084
	2:00 p.m.	Blood sample	0.079
10/17/23	8:00 a.m.	Fasting blood control	0.10
	8:05 a.m.	10 units insulin, H10 99 gm. glucose by mouth, 248 c.c. water	
	9:00 a.m.	Blood sample	0.127
	10:00 a.m.	Blood sample	0.086
	11:00 a m.	Blood sample	0.087
	12:00 m.	Blood sample	0.090
	1:00 p.m.	Blood sample	0.073
	2:00 p.m.	Blood sample	0.082

* Urine: Two samples during test gave a ++ sugar reaction
† Urine: One sample toward the end of the test gave a + sugar reaction
‡ Urine: Two samples during the test were sugar free.

Comment.—Chart 1 makes the effect of insulin in this patient's glucose absorption utilization tests more clear. Two things in particular are brought out in addition to the characteristic curve of the depressed phase of manic-depressive psychosis.

When insulin is given to the patient one-half hour before, or at the time of, the ingestion of glucose, the absorption seems to reach its height more quickly than when no insulin is given, but the amount of sugar present in the blood at this time is not as great as the highest amount found when no insulin was given. However, it is definitely higher than the control in point of time.

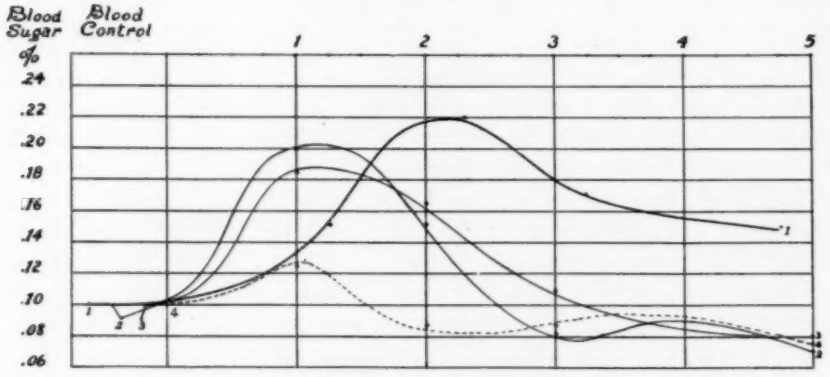

Chart 1 (Case 1).—Typical simple depression of severe type. Curve 1 indicates the blood sugar control observations without administration of insulin. Two samples of urine during the test gave a two plus sugar reaction. Curve 2: Glucose curve after the administration of 2 units of insulin (Lilly H10) subcutaneously thirty minutes before 99 gm. of glucose were given. One urine sample toward the end of the test gave a one plus sugar reaction. Curve 3: Glucose curve after the administration of 2 units of insulin subcutaneously at the same time that 99 gm. of glucose were given. Examination of two samples of urine was negative. Curve 4: Curve after the administration of 10 units of insulin subcutaneously at the same time that 99 gm. of glucose were given. This amount of insulin brings the curve to normal or slightly below. No urine test was made.

When insulin is given, the utilization of glucose is much more rapid. Depending on the amount of insulin given, it conforms to the utilization curve of normal persons. It may be said that the amount of insulin necessary to bring the curve to that of a normal person, who has not a case of depression, represents the degree of dysfunction of the chemical processes involved in the absorption and utilization of glucose in the body in this person.

All observations were made at a time when this patient was in a depressive phase.

CASE 2.—Susan D., a white woman, aged 52, married, was admitted to the State Psychopathic Hospital, July 9, 1923, with a diagnosis of manic-depressive psychosis, agitated depression, second attack, occurring in the involutional period. The family history appeared to be essentially negative, except that one sister was diabetic, and there was a suggestion of cycloid temperament type in the mother. The past personal history showed two previous attacks of mental disorder, one at the age of 35, the exact type of which was probably manic-depressive; the other in association with the menopause at 49, which was similar to the present difficulty and had its onset in June, 1923.

TABLE 2.—*Glucose Absorption Utilization Observations in Case 2*

Without Insulin

Date	Time		Blood Sugar Percentage
7/14/23*	8:15 a.m.	Fasting blood control	0.123
	8:20 a.m.	87 gm. glucose, 218 c.c. water	
	9:23 a.m.	Blood sample	0.200
	10:23 a.m.	Blood sample	0.149
	11:23 a.m.	Blood sample	0.159
	12:23 p.m.	Blood sample	0.142
	1:23 p.m.	Blood sample	0.102

With Insulin

7/26/23†	8:15 a.m.	Fasting blood control	0.107
	8:17 a.m.	2 units insulin, H10, subcutaneously 87 gm. glucose by mouth, 218 c.c. water	
	9:00 a.m.	Blood sample	0.095
	10:00 a.m.	Blood sample	0.106
	11:00 a.m.	Blood sample	0.130
	12:00 a.m.	Blood sample	0.112
	1:00 p.m.	Blood sample	0.093
	2:00 p.m.	Blood sample	0.087

* Urine: Three samples during test were sugar free.
† Urine: Two samples during test and twenty-four hour sample were sugar free.

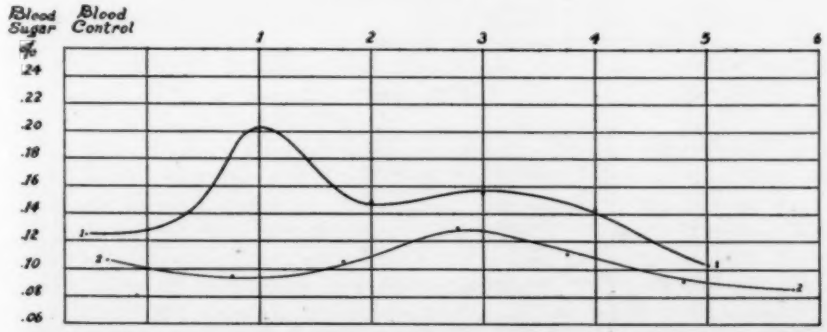

Chart 2 (Case 2).—Agitated depression, involutional type, second attack. The test was made in the absence of agitational reactions, the patient being depressed. Curve 1 is the control glucose curve without the administration of insulin. Three specimens of urine were sugar free. Curve 2: Two units of insulin subcutaneously at the same time that 87 gm. of glucose were given, brings the curve to normal. Two specimens of urine during the test and a twenty-four hour sample were sugar free.

On examination at the State Psychopathic Hospital, this was found to be a depression typical of the manic-depressive type. The patient was profoundly depressed in attitude and expression, tearful, with episodes of agitation. She frequently refused food, had suicidal tendencies and entertained numerous delusional ideas of a depressional type; thus ideas of personal sin, unworthiness, and impending misfortune were present. Physical, neurologic and routine laboratory examinations were negative. The patient weighed 49.6 kg. Her condition continued as described without improvement until her discharge from the hospital, Aug. 14, 1923. The tests herein recorded were made during a depressional phase.

Comment.—It will be observed that it required very much less insulin to overcome the depressive curve in this case than in the previous case. The depression in this patient was much less intense (Chart 2).

CASE 3 (Chart 3).—Bridget G., a white woman, aged 48, married, was admitted to the State Psychopathic Hospital, June 16, 1923, with a diagnosis of manic-depressive psychoses, agitated depression, occurring during the involutional period. The family history appeared to be essentially negative except that two sisters of the patient were described as being high-strung, nervous and of an excitable type. The previous personal history appeared to be negative up to the onset of the present trouble, which was first noted in February, 1923. On examination at the State Psychopathic Hospital, the disturbance was determined to be

TABLE 3.—*Glucose Absorption Utilization Observations in Case 3*

Date	Time		Blood Sugar Percentage
		Without Insulin	
7/13/23*	8:15 a.m.	Fasting blood control	0.095
	8:20 a.m.	94 gm. glucose by mouth, 235 c.c. water	
	9:15 a.m.	Blood sample	0.219
	10:15 a.m.	Blood sample	0.173
	11:15 a.m.	Blood sample	0.178
	12:15 p.m.	Blood sample	0.116
	1:15 p.m.	Blood sample	0.087
		With Insulin	
7/26/23†	8:15 a.m.	Fasting blood control	0.097
	8:17 a.m.	2 units insulin, H10, subcutaneously 94 gm. glucose, 235 c.c. water	
	9:00 a.m.	Blood sample	0.159
	10:00 a.m.	Blood sample	0.202
	11:00 a.m.	Blood sample	0.180
	12:00 m.	Blood sample	0.130
	1:00 p.m.	Blood sample	0.127
	2:00 p.m.	Blood sample	0.110
10/17/23	8:15 a.m.	Fasting blood control	0.10
	8:20 a.m.	10 units insulin, H10, subcutaneously 94 gm. glucose, 235 c.c. water	
	9:15 a.m.	Blood sample	0.11
	11:15 a.m.	Blood sample	0.15
	12:15 p.m.	Blood sample	0.073
	1:15 p.m.	Blood sample	0.068
	2:15 p.m.	Blood sample	0.080

* Urine: Two samples during test and twenty-four hour sample were sugar free.
† Urine: Sample near end of test was sugar free.

a depression typical of the manic-depressive type. There was marked depression of affect, frequent weeping and refusal of food, associated agitational reactions, depressional delusions and suicidal trends. Physical, neurologic and routine laboratory examinations were negative. She weighed 53.5 kg. The patient's condition continued as described without improvement until her transfer to the St. Joseph's Retreat at Dearborn, Mich., Oct. 23, 1923. The tests herein recorded were made during a depressional phase.

Comment.—This patient, as in Case 1, was in a phase of marked depression during the time these observations were made. This is well shown in Chart 3. Ten units of insulin were not sufficient to bring the curve to normal. It differs from the usual curve in a case of the depressed phase of manic-depressive psychosis in that it still shows the characteristic delayed utilization of carbohydrate.

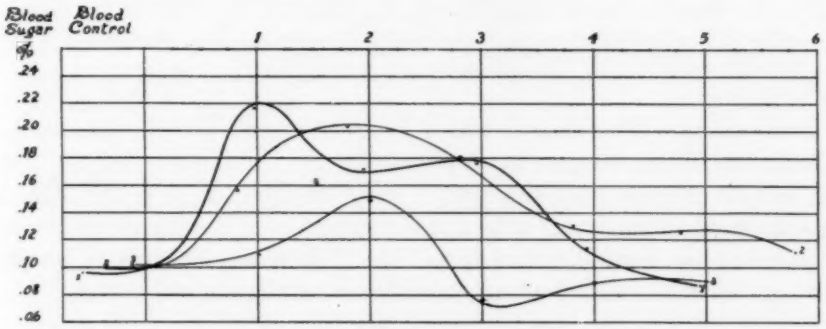

Chart 3 (Case 3).—Agitated depression, involutional type. The test was made during the depressive phase. Curve 1 is the glucose curve without the administration of insulin. It is a typical depressive curve. Two specimens of urine and a twenty-four hour specimen were sugar free. Curve 2: Glucose curve after 2 units of insulin were given subcutaneously at the same time that 94 gm. of glucose were given. This had little or no effect on the depressive curve. Urine specimen at the end of the test was sugar free. Curve 3: After 10 units of insulin had been administered. This was not quite sufficient to bring the curve to normal.

Case 4.—Herbert C., a white man, aged 37, married, was admitted to the State Psychopathic Hospital, June 20, 1923, with a diagnosis of manic-depressive psychosis, agitated depression, first attack. The family history showed marked alcoholic and psychotic stigmatization. The previous personal history was essentially negative except for indication of alcoholism, which may possibly be construed to be of significance as a manic equivalent. The present trouble dated from June, 1923, when the patient severely injured his left eye. Because of this, he became greatly depressed and discouraged. Examination at the State Psychopathic Hospital showed the situation to be one of depression of the manic-depressive type, with marked affective depression, thought difficulty, psychomotor retardation, suicidal trends, episodes of agitation, frequent refusal of food and associated delusions of personal sin, unworthiness and impending misfortune. Physical, neurologic and routine laboratory examinations were negative. He weighed .73.2 kg. The patient's condition continued as described without improvement until his discharge from the hospital, July 31, 1923. The observations on his carbohydrate utilization were made during a depressive phase.

TABLE 4.—*Glucose Absorption Utilization Observations in Case 4*

Date	Time		Blood Sugar Percentage
		Without Insulin	
7/25/23*	8:33 a.m.	Fasting blood control	0.110
	8:35 a.m.	128 gm. glucose, 320 c.c. water	
	10:15 a.m.	Blood sample	0.245
	11:15 a.m.	Blood sample	0.175
	12:15 p.m.	Blood sample	0.094
	1:15 p.m.	Blood sample	0.075
	2:15 p.m.	Blood sample	0.074
		With Insulin	
7/28/23†	8:00 a.m.	Fasting blood control	0.110
	8:10 a.m.	2 units insulin, H10, subcutaneously 128 gm. glucose, 320 c.c. water	
	9:10 a.m.	Blood sample	0.118
	10:00 a.m.	Blood sample	0.115
	11:00 a.m.	Blood sample	0.100
	12:00 m.	Blood sample	0.094
	1:00 p.m.	Blood sample	0.090
	2:00 p.m.	Blood sample	0.085

* Urine: One sample during test gave a + sugar reaction.
† Urine: Samples during test were sugar free.

Comment.—This patient was a large man with a thick panniculus. According to his weight, he required 128 gm. of glucose for his test. It will be observed that the glucose curve in this patient is the highest encountered in the series although it required only 2 units of insulin to make this flat, quite different from Case 1, in which 10 units of insulin did not have as marked an effect on lowering the glucose curve.

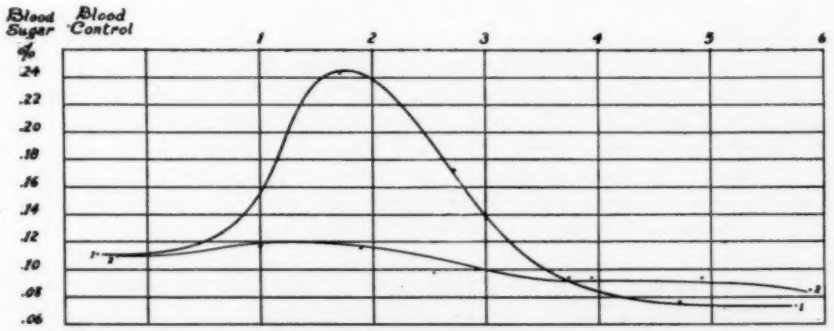

Chart 4 (Case 4).—Typical depressions with agitational features. The test was made in the absence of agitational reactions, the patient being depressed. Curve 1 is the control curve without the administration of insulin. One specimen of urine during the test gave a one plus sugar reaction. Curve 2: After 2 units of insulin had been administered at the same time as 128 gm. of glucose. This flattened out the curve.

CASE 5.—Beulah H., a white woman, aged 28, weighing 42.2 kg., married, was admitted to the State Psychopathic Hospital, July 17, 1923, with a diagnosis of manic-depressive psychosis, depressed phase, first attack. The family history showed that the father had been heavily alcoholic and one brother had been of cycloid constitution. The previous personal history was negative aside from an indication of sexual irregularity prior to her marriage, with the contraction of a

Neisserian infection. There was a history of gastric difficulty some years before, which was thought to be of "ulcerous" nature. The present illness began in April, 1923, and was marked by affective depression, thought difficulty, pronounced psychomotor retardation, occasional agitated reactions, associated with numerous delusions of a typical depressional type and suicidal trends. Examination at the State Psychopathic Hospital showed the psychiatric status to be a depression of the manic-depressive type, substantially as described in the foregoing. Routine physical, neurologic and laboratory examinations were negative with the exception of an indication of a certain undernutrition. The patient's course continued as described without improvement until her discharge from the hospital, Oct. 16, 1923.

Comment.—If one should attempt to judge the degree of depression in this patient from the character of the initial control curve, he might easily pronounce it marked; while, if on the other hand, he should judge it from the amount of insulin that was necessary to bring the curve to normal or flatten it out, he might pronounce it of moderate degree as compared with that shown in Case 1.

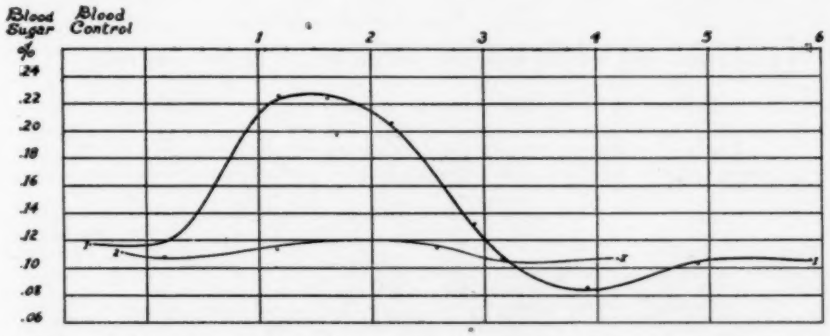

Chart 5 (Case 5).—Typical depression. Curve 1 is the control curve without insulin after 74 gm. of glucose were administered. The sugar reaction of the urine at the end of the test was two plus. Curve 2: After 2 units of insulin had been administered at the same time that 74 gm. of glucose were given. This flattened out the curve. The urine was sugar free after the test.

Case 6.—William D., a white man, aged 28, married, was admitted to the State Psychopathic Hospital, Dec. 14, 1922, with a diagnosis of manic-depressive psychosis, depressed phase, fifth attack. The family history showed a marked ancestral manic-depressive taint, and the patient himself appeared to be of a typical cycloid constitutional type with a history of four previous psychotic episodes, all of which were of a depressional type. The present difficulty was determined on examination at the State Psychopathic Hospital to be classic depression of the manic-depressive type with marked affective depression, thought difficulty, psychomotor retardation, occasional episodes of agitation, suicidal trends, refusal of food, stupor reactions and numerous and strongly marked delusions of a depressional nature. Somatically, aside from a certain undernutrition, no deviation was determined. The patient weighed 68 kg. Neurologic

and routine laboratory examinations were entirely negative. The patient's course continued as described without improvement until his transfer, Aug. 15, 1923, to the Pontiac State Hospital.

TABLE 5.—*Glucose Absorption Utilization Observations in Case 5 (Weight, 42.2 kg.).*

Date	Time		Blood Sugar Percentage
		Without Insulin	
7/31/23*	8:00 a.m.	Fasting blood control	0.117
	8:10 a.m.	74 gm. glucose and 178 c.c. water	
	9:30 a.m.	Blood sample	0.223
	10:30 a.m.	Blood sample	0.207
	11:00 a.m.	Blood sample	0.134
	12:00 m.	Blood sample	0.085
	1:00 p.m.	Blood sample	0.102
	2:00 p.m.	Blood sample	0.106
		With Insulin	
8/ 6/23†	8:30 a.m.	Fasting blood control	0.112
	8:40 a.m.	2 units insulin, H10, subcutaneously 74 gm. glucose, 178 c.c. water	
	9:00 a.m.	Blood sample	0.108
	10:00 a.m.	Blood sample	0.113
	11:15 a.m.	Blood sample	0.116
	12:00 m.	Blood sample	0.104
	2:00 p.m.	Blood sample	0.106

* Urine sample at end of test gave a ++ sugar reaction.
† Urine sample after test was sugar free. Patient did not urinate during test.

TABLE 6.—*Glucose Absorption Utilization Observations in Case 6*

Date	Time		Blood Sugar Percentage
		Without Insulin	
7/14/23*	8:00 a.m.	Fasting blood control	0.093
	8:02 a.m.	119 gm. glucose, 298 c.c. water	
	9:00 a.m.	Blood sample	0.166
	10:00 a.m.	Blood sample	0.170+
	11:00 a.m.	Blood sample	0.090
	12:00 m.	Blood sample	0.082
	1:00 p.m.	Blood sample	0.085
	2:00 p.m.	Blood sample	0.095
		With Insulin	
7/20/23†	8:00 a.m.	Fasting blood control	0.095
	8:15 a.m.	Two units insulin 119 gm. glucose, 298 c.c. water	
	9:00 a.m.	Blood sample	0.095
	10:00 a.m.	Blood sample	0.118
	11:00 a.m.	Blood sample	0.116
	12:00 m.	Blood sample	0.074
	1:15 p.m.	Blood sample	0.081
	2:00 p.m.	Blood sample	0.081

* Urine samples during test and twenty-four hour urine were sugar free.
† Urine samples during test and twenty-four hour urine were negative.

Comment.—No chart accompanies this case. It follows the same course as the others. Two units of insulin were sufficient to flatten out the curve. The control curve, on the other hand, is almost a normal curve, indicating that this patient's depression was not so profound as was suspected.

The ordinary method of determining the degree of depression in a patient is purely one of the personal equation of the examiner and the objective reaction of the patient. A patient wrings his hands and walks about possibly in an excited manner. We note in such a case moderate or marked agitational reaction. He sits quietly in the corner for indefinite periods of time. We record the condition as one of depression. Such a method of recording important data of this kind for comparative observations may not be entirely trustworthy. It does not and it cannot take into consideration the degree of mental activity, work, worry, etc., that is going on. It may be that the degree of prolongation of a glucose utilization curve, its height, and particularly the amount of insulin necessary to bring the curve to normal, is a more exact way of measuring the degree of depression for experimental purposes, since we know that during the agitational phase of this same form of cyclothymia the carbohydrate is utilized normally or more rapidly than normal.

CASE 7.—E. M. S., a white woman, aged 35, divorced, was admitted to the State Psychopathic Hospital, Sept. 14, 1923, with a diagnosis of manic-depressive insanity, depressed phase, first attack. The family history showed that the maternal grandmother had hung herself while in an attack of depression, with paranoid trends. One half-aunt was a drug addict. There was a record of an attack of a mental disorder two years before, which basically was probably of the manic-depressive type but which was associated with marked paranoid developments. The present trouble had its onset in August, 1923, with marked affective depression, thought difficulty, psychomotor retardation, delusions of a depressional character and suicidal trends. These findings were substantiated at examination on admission to the State Psychopathic Hospital. Physical examination revealed no deviation, except evidence of undernutrition. The patient weighed 39.1 kg. Neurologic and routine laboratory examinations were negative. Her hospital course showed marked accentuation of all features as described with recent evidence of the development of a typical depressional stupor with negativism and refusal of food.

TABLE 7.—*Glucose Absorption Utilization Observations in Case 7*

Date	Time		Blood Sugar Percentage
		Without Insulin	
10/31/23	8:00 a.m.	Control fasting blood	0.100
		58.5 gm. glucose, 146 c.c. water	
	9:00 a.m.	Blood sample	0.182
	10:00 a.m.	Blood sample	0.183
	11:00 a.m.	Blood sample	0.110
	12:00 m.	Blood sample	0.093
	1:00 p.m.	Blood sample	0.080
	2:00 p.m.	Blood sample	0.083
		With Insulin	
10/25/23	8:20 a.m.	Control fasting blood	0.103
	8:25 a.m.	58.5 gm. glucose, water 146 c.c.	
	9:20 a.m.	Blood sample	0.220
	9:25 a.m.	10 units insulin, H10, subcutaneously	
	10:20 a.m.	Blood sample	0.180
	11:20 a.m.	Blood sample	0.170
	12:20 p.m.	Blood sample	0.154
	1:20 p.m.	Blood sample	0.075
	2:20 p.m.	Blood sample	0.077

Comment.—The second observation on this patient does not follow the normal order of our previous investigations in that the insulin was not given until one hour after the glucose—hence the delay in the utilization is greater. A blood sample taken fifty-five minutes after the administration of glucose and five minutes before the administration of insulin showed rapid absorption. Instead of the curve dropping as it does in the control observation without insulin, it is prolonged two hours; in other words, ten units of insulin required a much longer time to bring the curve to normal than in our previous cases.

FURTHER CONTROLS

In the foregoing records, we have noted as controls the blood sugar curve of the depressed phase of manic-depressive psychosis without insulin. These are necessary in order to show any change induced by

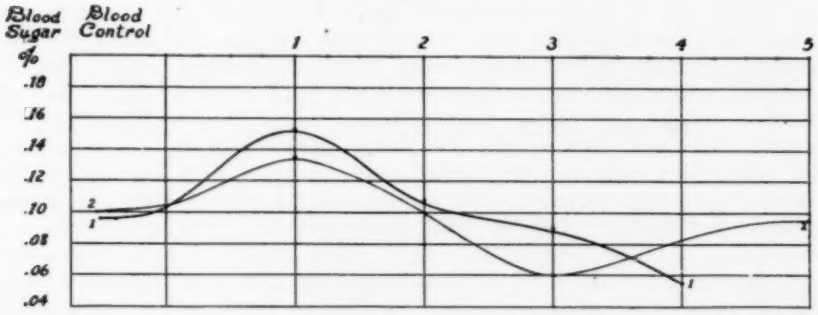

Chart 6.—These curves resulted from the tests made on a normal person. Curve 1 is the blood sugar curve that resulted when insulin was not administered. Curve 2 resulted when 5 units of insulin were given at the same time that 127.3 gm. of glucose were given. This caused only a slight lowering of the curve.

TABLE 8.—*Glucose Absorption Utilization Observations in a Normal Person* *

Date	Time		Blood Sugar Percentage
		Without Insulin	
2/11/24	8:50 a.m.	Fasting blood control	0.098
	8:55 a.m.	127.3 gm. glucose, 318 c.c. water	
	9:55 a.m.	Blood sample	0.154
	10:55 a.m.	Blood sample	0.105
	11:55 a.m.	Blood sample	0.087
	12:55 p.m.	Blood sample	0.055
		With Insulin	
2/14/24	8:30 a.m.	Fasting blood control	0.100
	8:35 a.m.	127.3 gm. glucose, 318 c.c. water	
	9:30 a.m.	5 units insulin subcutaneously	0.135
	10:30 a.m.	Blood sample	0.101
	11:30 a.m.	Blood sample	0.060
	12:30 p.m.	Blood sample	0.081
	1:30 p.m.	Blood sample	0.095

* Weight, 72.2 kg.

insulin but are not sufficient. We have made several similar observations on normal men—one of which is given as an example (Chart 6). It will be seen that the normal control curve is little influenced by a comparatively large dose of insulin, 5 units. We were prompted to use the larger dose as no change had occurred with the smaller amounts used in the depressive cases.

This patient, aged 19, had had a mild attack of scarlet fever and the temperature had returned to normal. There was still slight evidence of desquamation. The blood and urine were negative (Chart 6, Table 8).

The normal control curve of this normal person was little, if at all, influenced by an amount of insulin larger than that necessary to bring about a marked flattening of the curve in many of the manic-depressive depressions. In a normal person in whom we used the usual dose given to those with depression (2 units), a slightly higher curve was obtained than without insulin.

Observations on the clinical effect of insulin in manic-depressive psychosis in the depressive phase thus far have revealed no improvement. However, our work has not been sufficiently controlled to make a positive statement at this time.

CONCLUSIONS

1. The characteristic glucose utilization curve of the depressed phase in manic-depressive psychosis is made to conform to that of a normal person or is completely flattened out, or is made to approximate that of the agitational phase by the subcutaneous injection of insulin.

2. The amount of insulin necessary to accomplish this varies with the clinical status of the patient.

3. There seems to be evidence that the degree of depression may be measured by the amount of insulin necessary to bring the glucose utilization curve to that of a normal person.

4. The amount of insulin necessary to bring the curve to normal is a measure of the factors opposing the utilization of glucose.

THE PROGNOSTIC VALUE OF ARTERIAL HYPERTENSION IN THE PSYCHOSES OF LATER LIFE*

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In the January, 1922, number of the *American Journal of Psychiatry,* there appeared an article by Drs. August Hoch and John T. McCurdy, entitled, "The Prognosis of Involutional Melancholia." In this paper, a series of sixty-seven cases was reported. These cases were grouped into recovering and nonrecovering classes, and an analysis was made of the psychiatric symptoms. The conclusion reached by these investigators was that recovery took place unless there existed marked insufficiency of affect, peevish or auto-erotic behavior, or ridiculous hypochondriacal delusions usually concerned with the alimentary tract.

In this report, I intend to question in no way the conclusions reached by Hoch and McCurdy, which I regard as valuable and reliable. Instead, however, of approaching the problem from the angle of the presenting psychiatric symptoms, I have studied a small group of cases in relation to a physiologic finding—the blood pressure.

METHOD

Patients over 40 years of age were studied. Those cases were first eliminated in which there were frank arteriosclerosis, nephritis, as evidenced by albumin in the urine, or a positive Wassermann reaction. The blood pressures in a residual group of twenty-seven cases were compared. It was recognized that single blood pressure readings are inconclusive, as a person's blood pressure varies with his posture, the time of day, emotional condition, etc. But these factors were disregarded as they applied equally, presumably, to all cases. The systolic blood pressure was then taken by the ordinary method with stethoscope, cuff, and sphygmomanometer. The average of the readings taken on one patient was considered the blood pressure of that person. The cases were then divided into recovering and nonrecovering groups, and the average blood pressures in the groups was determined. The findings are shown in Table 1.

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* Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June, 1924.

TABLE 1.—*Average Blood Pressure in Groups Studied*

	Recovering Cases (19)	Non-Recovering Cases (8)
Males	133 (12)	181 (3)
Females	127 (7)	189 (5)
Average	131	187

It is to be noted that among the recovering cases the blood pressure of the males is higher than that of the females, as is found among normal persons. Among the nonrecovering cases, however, the blood pressure of the males is lower than that of the females.

Of course, the first question to be considered is the age factor. In Table 2 the average ages of the patients are shown.

TABLE 2.—*Average Ages of Patients Studied*

	Recovering Cases (19)	Non-Recovering Cases (8)
Males	57 (12)	56 (3)
Females	60- (7)	60 (5)
Average	58	58½

An average difference of one-half year is presumably not sufficient to account for an average difference of 56 points in blood pressure.

COMMENT

From such a limited group of cases, inferences can be made, but no conclusions drawn. So far as the evidence herewith presented is concerned, it seems that the prognosis of the psychoses of the later years is poor if hypertension is present to complicate the situation, but good if hypertension is not found.

Data are being gathered to determine whether the assumption is justified that in a given case the prognosis improves as the blood pressure falls, and grows more grave as the blood pressure rises.

Perhaps hypertension in a psychotic patient merely means that the stage is set for the development of arteriosclerosis. If we knew the cause of arterial hypertension, the situation would be clearer. Some observers consider gonadal dysfunction a cause of hypertension, and claim that the administration of gonadal substances by mouth or by grafting reduces hypertension. Be that as it may, the term involutional psychosis implies a psychosis dependent on an endocrine maladjustment. Physiologic chemists often find in gonadal dysfunction, a

frankly lowered sugar tolerance, a lowered basal metabolism, a lowered carbon dioxid tension in the blood, and an increase of residual nitrogen in the urine.

The use of the term "involutional psychosis" seems to me unjustified unless it is supported by evidence of physiologic abnormality.

Among the psychoses of the later years are to be found cases of four general types: simple functional cases without evident physiologic abnormality; functional cases with transient physiologic abnormality; cases with definite physiologic abnormality which probably later become the cases that comprise the fourth group—cases with definite pathologic conditions. The prognosis in the first type is good; the prognosis in the last group is bad; the prognosis in the intermediate groups is indefinite, but should be made more definite by careful observation of the physiologic activities.

THE PROTEIN AND CELLULAR CONTENT OF THE CEREBROSPINAL FLUID

CHANGES IN THE FLUID FOLLOWING INTRASPINAL, INTRACISTERN AND INTRAVENTRICULAR THERAPY WITH SWIFT-ELLIS SERUM *

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A marked increase in the total protein and in the number of cells in the cerebrospinal fluid was observed after the administration of Swift-Ellis serum by the intraspinal, intracistern or intraventricular route. Cerebrospinal fluids were studied with especial reference to these constituents in twenty-five cases in which treatment was administered by the intraspinal route, in twenty-one cases in which intracistern therapy had been given, and in five cases in which intraventricular treatment had been used. These patients had general paralysis, tabes dorsalis and cerebrospinal syphilis.

The method used in the protein determinations was that described by Denis and Ayer [1] in 1920, and the normal standard was that of Ayer and Foster,[2] who consider the limits of normal as being between 16 and 38 mg. per 100 c.c. and any total protein quantitation above 40 mg. as distinctly pathologic.

EFFECT ON LUMBAR SPINAL FLUID OF LUMBAR SUBARACHNOID INJECTION

Twenty-four hours after the injection of Swift-Ellis serum in the lumbar region in quantities varying from 10 to 20 c.c., we found that the cells and total protein of the fluid in this region were very much increased. This increase was present after forty-eight hours, and after six or seven days had entirely disappeared. Table 1 shows these observations. Three cases showed an increase of 12, 180 and 195 cells twenty-four hours after the injection of the serum. Of four cases

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* From the Boston Psychopathic Hospital.

* This investigation was aided by funds from the Division of Mental Hygiene of the Massachusetts Department of Mental Diseases.

1. Denis, W., and Ayer, J. B.: A Method for the Quantitative Determination of Protein in the Cerebrospinal Fluid, Arch. Int. Med. **26**:436 (Oct.) 1920.

2. Ayer, J. B., and Foster, H. E.: Quantitative Estimation of Total Protein in Spinal Fluid, J. A. M. A. **77**:365 (July 30) 1921.

observed forty-eight hours after serum was injected, three showed an elevation of 23, 34 and 10 cells above their original level, and one showed the cells to be practically back at their initial level. In one observation after six days, there was an increase of six cells above the initial level. Eleven observations taken seven days after the injection of serum showed the cellular content to be approximately the same as before the injection, while in one, the cell count was 30 lower, and in another it was 15 cells higher than prior to the injection. On the whole, however, one may say that seven days after an intraspinal injection of Swift-Ellis serum, the cell count was about the same as before the serum was introduced.

TABLE 1.—*Effect on Total Protein and Cells of Lumbar Spinal Fluid at Varying Intervals Following Lumbar Injection of Swift-Ellis Serum*

No.	Intervals After Injection	Cells: Lumbar Fluid			Total Protein: Lumbar Fluid			Diagnosis
		Before	After	Difference	Before	After	Difference	
1	24 hours	36	48	+ 12	93.4	140.9	+ 47.5	General paralysis
2	24 hours	10	205	+195	73.6	209.4	+135.8	General paralysis
3	24 hours	8	188	+180	62.0	234.8	+172.8	General paralysis
4	48 hours	13	36	+ 23	255.0	309.0	+ 54.0	General paralysis
5	48 hours	12	46	+ 34	62.0	119.1	+ 57.1	General paralysis
6	48 hours	32	28	— 5	168.4	336.9	+168.5	General paralysis
7	48 hours	20	30	+ 10	143.5	168.0	+ 24.5	General paralysis
8	6 days	12	18	+ 6	86.1	89.7	+ 3.6	General paralysis
9	7 days	44	32	— 12	113.9	110.7	— 3.2	General paralysis
10	7 days	36	6	— 30	85.1	86.1	+ 1.0	General paralysis
11	7 days	40	42	+ 2	72.5	77.5	+ 5.0	General paralysis
12	7 days	3	5	+ 2	40.2	40.2	0	Tabes with optic atrophy
13	7 days	8	10	+ 2	43.1	41.2	— 1.9	Tabes with optic atrophy
14	7 days	9	12	+ 3	41.3	37.7	— 3.6	Tabes with optic atrophy
15	7 days	10	9	— 1	49.0	39.4	— 9.6	Tabes with optic atrophy
16	7 days	8	6	— 2	37.2	36.9	— 0.3	Tabes with optic atrophy
17	7 days	8	6	— 2	67.4	64.1	— 3.3	General paralysis
18	7 days	1	16	+ 15	40.8	69.2	+ 28.4	General paralysis
19	7 days	8	8	0	69.2	62.0	— 7.2	General paralysis

Similar observations were made with regard to the total protein. Twenty-four hours after the administration of serum intraspinally, the total protein of the lumbar spinal fluid was found to be increased 47 mg., 136 mg. and 172 mg., respectively, in three cases. At the end of forty-eight hours, the protein was still greatly increased, in amounts fully as great as at the end of twenty-four hours, four cases showing increases of 25, 54, 57 and 168 mg. after the injection of Swift-Ellis serum. One observation taken at the end of six days shows the total protein practically at its initial level. Eleven observations were taken seven days after the injection of serum. In nine cases, the total protein was practically the same in amount as before the injection of serum. Of the two remaining cases, one showed, after seven days, a drop of 10 mg. below the original level, and the other was 29 mg. above the initial level. The latter, interestingly enough, occurred in the case in which the cells were still elevated above their original level after seven days.

EFFECT ON LUMBAR FLUID OF CISTERN INJECTION OF SWIFT-ELLIS SERUM

A similar rise in the cells and total protein of the lumbar spinal fluid follows an injection of Swift-Ellis serum into the cisterna magna. Table 2 illustrates this point.

In this series of experiments, a cistern puncture was performed, cistern fluid removed and Swift-Ellis serum administered by the cistern route. The cell count and total protein determinations were made on the cistern fluid. At the end of twenty-four hours, a lumbar puncture

TABLE 2.—*Effect on Total Protein and Cells of Lumbar Spinal Fluid at Varying Intervals Following Intracisternal Injection of Swift-Ellis Serum*

		Cells				Total Protein				
		Cistern Fluid	Lumbar Fluid			Cistern Fluid	Lumbar Fluid			
No.	Intervals After Injection	Before	Before	After	Difference	Before	Before	After	Difference	Diagnosis
1	24 hours	11	18	25	+ 7	60.5	73.1	172.5	+ 99.4	General paralysis
2	24 hours	7	8	11	+ 3	69.2	73.4	164.9	+ 91.5	General paralysis
3	24 hours	9	17	39	+ 22	70.4	73.1	127.9	+ 54.8	General paralysis
4	24 hours	3	4	4 *	0	40.2	73.4	168.0	+ 94.6	General paralysis
5	24 hours	25	36	323	+ 187	50.0	71.7	168.0	+ 96.3	General paralysis
6	24 hours	18	18	18	0	65.1	85.2	138.5	+ 53.3	General paralysis
7	24 hours	12	36	1,710	+1,674	53.1	71.7	151.9	+ 80.2	General paralysis
8	24 hours	32	35	313	+ 278	127.3	155.0	369.0	+214	General paralysis
9	24 hours	59	36	31	— 5	287.4	100.6	369.0	+268.4	General paralysis
10	24 hours	5	12	340	+ 328	31.5	62.0	193.7	+131.7	General paralysis
11	24 hours	10	12	228	+ 216	45.6	62.0	109.2	+ 47.2	General paralysis
12	24 hours	3	33	*	.....	77.5	101.9	104.7	+ 2.8	General paralysis
13	48 hours	43	33	26	— 7	40.1	101.9	107.6	+ 5.7	General paralysis
14	48 hours	23	26	32	— 6	310.0	310.0	369.0	+ 59.0	General paralysis
15	48 hours	48	36	43	+ 7	125.0	100.6	193.7	+ 93.1	General paralysis
16	48 hours	38	36	258	+ 222	123.0	100.6	215.2	+114.6	General paralysis
17	48 hours	30	36	40	+ 4	71.7	71.7	276.8	+205.1	General paralysis
18	48 hours	39	36	1,150	+1,414	161.4	*	337.0	.....	General paralysis
19	3 days	8	1,633	*	.....	53.4	66.2	65.7	— 0.5	Neurosyphilis
20	4 days	48	33	26	— 7	94.5	101.9	123.0	+ 21.1	General paralysis
21	7 days	12	16	8	— 8	49.7	66.2	53.4	— 12.8	Neurosyphilis

* Cells and protein not determined.

was performed, and the cell count and total protein determinations were made on this. Since lumbar puncture was not performed at the time of the cistern puncture because of the discomfort to the patient, we have taken as figures for comparison of the lumbar cells and protein before and after the cistern puncture, either determinations made on these constituents of the lumbar fluid the day previously, or average figures which represent several determinations made on the spinal fluid without the administration of serum, so that in Table 2 one finds the cells and total protein of the cistern fluid and of the lumbar fluid before and after the injection of the serum. The same explanation holds true of Tables 3, 4 and 5.

Of eleven observations taken at the end of twenty-four hours after the cistern injection, eight cases showed a rise in the cells, varying from a slight to a marked increase, and of these eight cases, three showed

an increase of from 3 to 22 cells, and five showed increases of 187, 1,674, 278, 328 and 216 cells. Of the remaining three cases, two showed no increase and one was decreased by 5 cells. At the end of forty-eight hours, as shown by observations on six cases, two showed a decrease in cells, while four showed increases of 7, 4, 222 and 1,114 cells. At the end of four days, the cells were practically at their initial levels, and the same was true at the end of seven days.

TABLE 3.—*Effect on Total Protein and Cells of Lumbar Spinal Fluid at Varying Intervals Following Intraventricular Injection of Swift-Ellis Serum*

		Cells				Total Protein				
		Ventricular	Lumbar Fluid			Ventricular	Lumbar Fluid			
No.	Intervals After Injection	Fluid Before	Before	After	Difference	Fluid Before	Before	After	Difference	Diagnosis
1	24 hours	8	20	46	+ 26	25.5	86.0	287.0	+201	General paralysis
2	24 hours	22	26	198	+172	14.7	70.2	219.0	+148.8	General paralysis
3	24 hours	22	32	51	+ 19	74.0	86.1	221.4	+135.3	General paralysis
4	6 days	0	31	16	— 15	21.0	91.2	89.0	+ 1.8	General paralysis
5	9 days	7	31	86	+ 55	14.8	91.2	116.1	+ 24.9	General paralysis

TABLE 4.—*Effect on Total Protein and Cells of Cistern Fluid at Varying Intervals Following Lumbar Injection of Swift-Ellis Serum*

		Cells				Total Protein				
		Lumbar	Cistern Fluid			Lumbar	Cistern Fluid			
No.	Intervals After Injection	Fluid Before	Before	After	Difference	Fluid Before	Before	After	Difference	Diagnosis
1	24 hours	31	5	42	+37	91.2	*	172.2	?	General paralysis
2	24 hours	13	3	18	+15	55.3	45.6	103.3	+57.7	General paralysis
3	48 hours	20	8	12	+ 4	110.7	53.4	49.7	— 6.3	Neurosyphilis
4	7 days	42	25	18	— 7	77.5	50.0	65.1	+15.1	General paralysis
5	14 days	12	*	10	?	50.4	*	49.9	?	General paralysis

* Cells and protein not determined.

TABLE 5.—*Effect on Total Protein and Cells of Intraventricular Fluid at Varying Intervals Following Lumbar Injection of Swift-Ellis Serum*

		Cells				Total Protein				
		Lumbar	Ventricular Fluid			Lumbar	Ventricular Fluid			
No.	Intervals After Injection	Fluid Before	Before	After	Difference	Fluid Before	Before	After	Difference	Diagnosis
1	24 hours	32	18	18	0	110.7	25.5	28.1	+2.6	General paralysis
2	24 hours	13	8	5	0.3	117.4	25.5	20.3	—5.2	General paralysis

The total protein was increased in the lumbar spinal fluid after the injection of Swift-Ellis serum into the cistern. At the end of twenty-four hours, eleven cases showed a marked increase in the protein of the lumbar fluid. The increase varied from 472 to 268.4 mg. per 100 c.c. One case showed an increase in the protein of 2.8 mg. per

100 c.c. At the end of forty-eight hours, there was still an increase in protein in amounts as high as after twenty-four hours. At the end of three days, the protein in the lumbar spinal fluid in one case was 5 mg. less than in the beginning. At the end of four days, it was increased 21.1 mg. in one case, and after seven days, had returned to even lower than its normal level.

EFFECT ON LUMBAR FLUID OF VENTRICULAR INJECTION OF SWIFT-ELLIS SERUM

Almost similar results are obtained in the lumbar spinal fluid if one introduces Swift-Ellis serum into the ventricles. Table 3 illustrates this point. In these experiments, Swift-Ellis serum was introduced into the ventricles, and observations were made on the lumbar spinal fluid at regular intervals. Three observations at the end of twenty-four hours after the introduction of serum into the ventricle showed the cells increased above their original level. At the end of six days, in one case there were 15 cells less than the original number, while at the end of nine days, in one case there was an increase of 55 cells in the lumbar spinal fluid over the previous number. The protein showed great increases after twenty-four hours, varying from 135.3 to 201 mg. per 100 c.c. At the end of six days, it was almost back to its original level in one case, and in another case after nine days was increased to the extent of 24.9 mg.

EFFECT ON CISTERN FLUID OF LUMBAR SUBARACHNOID INJECTION OF SWIFT-ELLIS SERUM

When Swift-Ellis serum was introduced into the lumbar region, there was an increase in the cells and total protein of the fluid in the cisterna magna after twenty-four hours. Two cases in which the cistern fluid was examined twenty-four hours after Swift-Ellis serum was given intraspinally showed an increase of 37 and 15 cells, and of 80 and 48 mg. of protein. At the end of forty-eight hours, the cells and protein were back to their original levels, the same holding true after seven and fourteen days. This would seem to indicate that a certain amount of the serum introduced intraspinally diffuses upward during the course of twenty-four hours, but only in small quantities, so that evidence of this diffusion is absent at the end of forty-eight hours.

EFFECT OF THE LUMBAR SUBARACHNOID INJECTION OF SWIFT-ELLIS SERUM ON THE VENTRICULAR FLUID

When Swift-Ellis serum was introduced by the intraspinal route, there was no increase in the cells or protein of the ventricular fluid in two cases at the end of twenty-four hours after the introduction of the serum.

CAUSE OF CELLULAR INCREASE

In most cases, the majority of the cells were polymorphonuclears, with a few small lymphocytes and some large lymphocytes. In some cases, however, the lymphocytes were more numerous. The cells probably owe their origin to the irritative effect of the serum on the meninges. Boveri [3] indirectly remarks on this when he states that in affections involving the meninges the leukocytes are generally increased. One can show, moreover, that the introduction of a salt solution into the spinal canal produces an increase in cells, which is probably due to the irritant effect of the salt solution on the meninges.

In three cases in which the patients were given physiologic sodium chlorid intraspinally, the cell count was increased from 4 to 458 and 650 in two of the cases after twenty-four hours, and from 4 to 420 forty-eight hours after the saline was administered in a third. Boyd [4] remarks that the cells in the perivascular spaces in general paralysis and sleeping sickness "have arisen from the fixed cells of the tissue in response to the action of a chronic irritant." Szecsi [5] found that irritants such as saline could increase the cells in the spinal fluid.

Whatever the origin of the cells—whether the blood or the tissues—it seems to be true that a chronic irritant is capable of increasing the cells in the cerebrospinal fluid, and that irritants such as serum and saline introduced into the spinal fluid may increase the cell count either by producing a hyperemia and calling cells from the blood stream, or by producing a direct, irritant effect on the meninges. The safer course would be to assume that the cells probably come from both sources.

CAUSE OF PROTEIN INCREASE

To find the source of the protein offers greater difficulties. All our observations were made on patients with general paralysis, tabes, or cerebrospinal syphilis. Numerous observers have pointed out the fact that the protein in the spinal fluid is greatly increased in these conditions, so that one may safely assume this to be the case. It is not the protein which is present as a result of the disease process, but that which one finds after the injection of serum, which we should like to explain. It is undoubtedly true that a large part of the increased protein which is observed is due to the protein in the serum which is introduced.

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3. Boveri, P.: Dissociation of Albumin and Leucocytes in Cerebrospinal Fluid, Policlin. **29**:1001, 1922.

4. Boyd, H.: The Cerebrospinal Fluid, New York, The Macmillan Company, 1920.

5. Szecsi, S.: Weitere Beiträge zur Cytologie der Liquor Cerebrospinalis, Ztschr. f. d. ges. Neurol. u. Psychiat. **60**:481-492, 1912.

But not all the protein comes from this source. Ayer and Foster feel that the spinal fluid protein in pathologic states has its origin from at least two sources: exudative and transudative, the protein coming from the blood, in the latter process, by an increased permeability of the membranes. Although the authors do not specify, the protein due to the exudative process is probably obtained from the leukocytes which accumulate in the spinal fluid as a result of meningeal irritation. This is seen in an acute pyogenic meningitis, and to a less extent in a syphilitic meningitis. But, as the authors point out, "within the syphilitic group itself there are subgroups which show slight cell reaction and relatively great protein content."

Mestrezat [6] doubts the value of cells as a source of origin of spinal fluid protein, and remarks on the rarity of the phenomenon of digestion and cytolysis in the ordinary meningeal irritations. Boveri [3] also points out that while in affections involving the meninges the increase in albumin in the cerebrospinal fluid usually parallels the increase in the number of leukocytes, in some conditions such as Pott's disease or compression from an intraspinal tumor, there is a divergent behavior of the albumin and leukocytes. Bickel,[7] in a study of the spinal fluid findings in tuberculous meningitis, finds the protein often increased along with the number of cells, but in the early stages of the disease he found the cells increased and the protein normal, while in the later stages the cells were occasionally few and the protein abundant. It would seem, therefore, that in many cases an increase in cell count and an increase in protein in the spinal fluid are concomitant, but that this is not always so, because one may have an increased protein without an increased cell count. There must, therefore, be another source of protein, and this may be exudative due to meningeal irritation. The source of the increased protein in these experiments would seem to be: (1) protein present as a result of the introduction of serum into the cerebrospinal fluid; (2) protein accompanying an increase in cells in the cerebrospinal fluid, either having its origin from the leukocytes themselves, or being present in increased amount because of its exudative origin.

AGGREGATION OF MATERIAL IN THE LUMBAR REGION AS COMPARED TO THE CISTERN AND VENTRICULAR REGIONS

In the experiments described above, Swift-Ellis serum introduced into the anterior horn of the lateral ventricles and into the cisterna magna was found in the lumbar spinal fluid after twenty-four hours,

6. Mestrezat, W.: Le liquide céphalo-rachidien, Paris, 1912.

7. Bickel, Georges: The Cerebrospinal Fluid in Tuberculous Meningitis, Arch. Suiss de Neurol. et Psychiat. **12**:269, 1923.

as shown both by the gross appearance of the spinal fluid and by evidence of the presence of the serum in an increased cell count and total protein content. It was found, moreover, that the serum introduced in this way could still be detected after forty-eight hours, but had disappeared at the end of seven days. Moreover, serum which had been introduced into the lumbar subarachnoid space was found to have reached the cisterna magna in small quantities at the end of twenty-four hours, but no evidence of its presence in the cistern fluid could be found after forty-eight hours. In this experiment also, the serum could be detected grossly in the cistern fluid at the end of twenty-four hours, but could not be detected after forty-eight hours. On the other hand, serum could not be detected in the ventricular fluid, either grossly or by the presence of increased cells and protein twenty-four hours after the injection of Swift-Ellis serum in the lumbar region. These observations would seem to indicate that there is a ready communication between the various loci in the subarachnoid space, and that a substance such as serum introduced in the cistern or ventricular region either diffuses downward rapidly, or is carried down into the lumbar region by a downward flow of spinal fluid, or settles down into the lumbar region because of its greater specific gravity. Solomon, Thompson, and Pfeiffer [8] showed by in vitro experiments that blood serum when mixed with spinal fluid seeks the lowest level, and they express the opinion that "we may therefore assume that when the blood serum is introduced into the subarachnoid space, it will tend to reach the lowest portion of the cerebrospinal fluid system by a slow diffusion process." They seem to favor the idea that "the movement of the introduced substances (into the spinal fluid) may depend either on circulation of the cerebrospinal fluid or, what is more probable, on a diffusion of the substances due to osmotic and specific gravity effects." These authors found that neutral phenolsulphonephthalein diffused rapidly through cerebrospinal fluid whether placed at the bottom or at the top of the column. Blood serum, however, had no tendency to diffuse upward. Our experiments, however, show that blood serum can be detected in the cistern fluid both grossly and by the presence of increased cells and protein twenty-four hours after the introduction of serum intraspinally. No serum was found later. It would seem, therefore, that blood serum does diffuse upward when introduced at the bottom of a column of cerebrospinal fluid, but that this diffusion is slow and slight, depending on the specific gravity of the serum and the position of the patient after the injection.

8. Solomon, H. C.; Thompson, L. J., and Pfeiffer, H. M.: Circulation of Phenolsulphonephthalein in the Cerebrospinal System **79**:1014 (Sept. 23) 1922.

LENGTH OF TIME FOR THE DISAPPEARANCE OF THE INCREASED AMOUNT OF CELLS AND TOTAL PROTEIN

Examination of the lumbar spinal fluid after the subarachnoid lumbar injection of serum showed an increase in the cell and the total protein content twenty-four and forty-eight hours later, which gradually returned to normal within seven days. After serum was injected into the cistern and ventricle, the cell and the total protein content was increased in the lumbar spinal fluid, but similarly returned to normal within from three to seven days.

A moderate rise in the cells and total protein was observed in the cisternal fluid after lumbar injection of serum, which increase disappeared in forty-eight hours.

HOW DO CELLS AND PROTEIN DISAPPEAR?

The absorption of cerebrospinal fluid, according to Weed,[9] is a twofold process, chiefly by a rapid drainage into the great dural sinuses and in small part a slow, intermittent escape into the lymphatic vessels.

In our experiments, we found foreign substances, cells and serum that disappeared from the subarachnoid space at the end of six or seven days. Dandy and Blackfan[10] have shown that substances in the subarachnoid space appear in the general circulation but slowly. Dixon and Halliburton[11] also have shown that physiologic salt solution when introduced into the subarachnoid space disappears rapidly, the rate of disappearance being dependent on the diffusibility of the injected substances. Such substances as nicotin and epinephrin disappear quickly.

Further evidence of the diffusibility of substances introduced into the subarachnoid space is offered by the observation of Dandy and Blackfan that phenolsulphonephthalein appears rapidly in the urine after being introduced into the subarachnoid space.

From the observations made in these experiments, it is debatable whether or not the increased amount of cells and total protein escape into the general circulation.

SUMMARY AND CONCLUSIONS

1. A study of the cellular and protein contents of the cerebrospinal fluid is presented in twenty-five cases in which intraspinal therapy was used with Swift-Ellis serum, in twenty-one cases in which the intra-

9. Weed, L. H.: The Cerebrospinal Fluid, Physiol. Rev. **11**:171, 1922.

10. Dandy, W. E., and Blackfan, K. D.: An Experimental Study of Internal Hydrocephalus, J. A. M. A. **61**:2216 (Dec. 20) 1913; Internal Hydrocephalus, J. Dis. Child. **8**:406 (Dec.) 1914.

11. Dixon, W. E., and Halliburton, W. D.: Rapidity of Absorption of Drugs Introduced into the Cerebrospinal Fluid, J. Physiol. **44**:7, 1912.

cistern route was used, and in five cases in which intraventricular treatment was given.

2. After the intraspinal injection of Swift-Ellis serum in the lumbar region, there occurs a marked rise in the cells and the total protein of the lumbar fluid after twenty-four hours, which is still present after forty-eight hours, and has disappeared at the end of six or seven days.

3. A similar rise in the cells and the total protein of the lumbar spinal fluid is observed after the injection of Swift-Ellis serum by the cisternal and ventricular routes.

4. A moderate rise in the cells and the total protein of the cisternal fluid is observed twenty-four hours after the injection of Swift-Ellis serum by the lumbar route. This increase disappears at the end of forty-eight hours.

5. There is no increase in the cells and the total protein of the ventricular fluid twenty-four hours after the injection of Swift-Ellis serum into the lumbar region.

6. The increase in cells—which are chiefly polymorphoneuclear leukocytes—is due to the irritative action of the serum on the meninges.

7. The source of the protein increase is chiefly from the serum introduced, which in turn causes a mild exudative reaction, thereby increasing the protein.

8. The increase in the cells and the total protein of the lumbar spinal fluid after the injection of Swift-Ellis serum in the cisterna magna and the ventricles is presented as evidence of the diffusion of the serum from above downward.

9. A certain amount of serum introduced into the lumbar region diffuses upward, as seen by the increase in cells and protein in the cisterna magna after twenty-four hours. There is no evidence from these experiments of any upward current or circulation of the spinal fluid, certainly not into the lateral ventricles. A slight diffusion upward, probably due to gravity, is believed to occur.

10. The mechanism by which the cells are caused to disappear from the fluid after two or three days is not evident.

Clinical and Occasional Notes

THE RECENT DEVELOPMENT OF METAPSYCHOLOGY

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The theoretical foundation of psychanalysis, which Freud has called metapsychology, seems to become more and more abstracted from the corresponding clinical work. An account of the present status of metapsychology may therefore take the form of a study of the gradual development of the main psychanalytic theories.

Metapsychology has three fundamental principles: 1. The topographical principle relates to the spatial arrangement of mental phenomena in the conscious, foreconscious and unconscious spheres. 2. The dynamic principle refers to the measure in which the various tendencies or contents are invested[1] with certain quantities of libido. 3. The economic principle has to do with the fact that the mental apparatus tends to keep the quantity of excitation as low as possible, or at least constant. This is one of the important aspects of the pleasure principle. "The pleasure principle is derived from the constancy principle" (Freud).

Pleasure and pain constitute one of the three great polarities governing mental life: (1) the *biologic* polarity of activity and passivity; (2) the *real* one of self and outer world, and (3) the *economic* one of pleasure and pain.

From its inception, the psychanalytic doctrine has undergone a development involving rather far-reaching changes. It seems convenient and not too schematic to distinguish in this development five different stages. Using provisional brief designations for these five stages, we may distinguish: (1) the stage of sexual trauma; (2) the stage of the extension of the concept of sexuality; (3) the stage of introduction of narcissism and of the egopsychology; (4) the stage inaugurated by Freud's paper, "Beyond the Pleasure Principle," 1920, and (5) the stage of the unconscious part of the ego introduced by Freud's latest book, "Das Ich und das Es," 1923.

STAGE OF SEXUAL TRAUMA OF PSYCHANALYTIC DOCTRINE

In the first stage, Freud saw in the sexual trauma the chief factor of the neurosis. He pointed to special circumscribed "contents" incompatible with the dominant tendencies of the mental life of the neurotic. The individual's defense against this content constituted the neurosis. With this view, he put himself in opposition to Breuer, who thought that only such images can become pathogenic which take their origin in "hypnoid states." In this stage Freud, as he has pointed out later, overrated the importance of the influence of seduction in infantile life.

STAGE OF EXTENSION OF CONCEPT OF SEXUALITY

In the second stage, Freud emphasized in contrast to the infantile sexual trauma his well-known wider conception of sexuality, which he has described in "Three Contributions to the Sexual Theory," 1905. He evolved "circumscribed

1. It is preferable to translate Freud's term libido Besetzung with libido investment instead of "charge."

perversion tendencies," and described their relation to the psychosexual constitution. His paper, "Character and Analerotism," 1908, belongs to this period. In view of the later development, it is noteworthy that in this stage (1906) the first psychanalytic interpretation of a paranoid development was attempted. Abraham, in his paper, "The Psychosexual Differences of Hysteria and Dementia Praecox," claimed that in hysteria the libido investment of objects is lacking.

STAGE OF NARCISSISM AND EGO PSYCHOLOGY

In the third stage, the important concept of narcissism was introduced by Freud ("Introduction of Narcissism," 1914). Narcissism had been described clinically by Näcke as a sexual perversion, in which the individual loves himself. Narcissistic libido is the libido turned toward the ego. The ego tendencies had not received much attention in psychanalysis before. This is explained by the fact pointed out especially clearly by Hitschman, that as psychanalysis started originally from the study of the transference neuroses, repression was postulated and the existence of a repressing principle, that is to say, the ego tendencies, was taken for granted; and in the beginning the attention of psychanalysts was turned toward the repressed material. However, in this third stage of metapsychology, Freud claimed that the ego itself could become a sexual object, that is to say, the ego could be invested with sexual libido. There are therefore two types of libido, object libido and ego libido. In a primitive state of libido division, in complete narcissism, the sexual libido and the ego instincts are united and inseparable in the self-satisfied ego. This complete narcissism occurs in intra-uterine life and recurs in sleep. On account of this attention bestowed on the ego, one might think of this stage of metapsychology as the stage of egopsychology. Freud gives as an analogy for object and ego libido a protozoon which sends out its pseudopodia to the environment (object libido) or draws them in to itself (ego libido). The most important new fact evolved in this stage of psychanalysis is the formulation of narcissistic regression. When the libido is withdrawn from the outer world and turned toward the individual himself, this is nothing else but a regression to a former state where the ego is naturally invested with this libido.

At this stage of psychanalysis, the scheme of classification of psychopathologic conditions was evolved, comprising three different groups: (1) Narcissistic psychoneuroses (i. e., psychoneuroses in which there occurs a narcissistic regression): dementia praecox; paranoia; manic-depressive insanity. (2) Transference psychoneuroses (or psychoneuroses in which the fact that a considerable quantity of object libido is "free" is the distinguishing feature): conversion hysteria; anxiety hysteria; compulsion neurosis. (3) "Actual" neuroses (i. e., physiologic rather than psychologic maladies and dependent mainly on some interference in the normal *physical* sex life): neurasthenia; anxiety neurosis; hypochondriasis.[2]

STAGE INAUGURATED BY FREUD'S "BEYOND THE PLEASURE PRINCIPLE"

We may speak of a fourth stage of psychanalysis inaugurated by Freud's book, "Beyond the Pleasure Principle," 1920. To a large extent this book contains, as Freud emphasizes, far-reaching speculations in which he indulged out of scientific curiosity, to see how far the original ideas might lead. Two important points of the psychanalytic doctrine are fundamentally changed in

2. At present, however, hypochondriasis is classed as one of the narcissistic neuroses.

this paper: first, the extent of application of the pleasure principle is restricted. The psychanalytic doctrine had postulated two fundamental principles of psychic processes, the pleasure principle and the reality principle. The sexual impulses are guided by the pleasure principle for the most part. If they cannot find immediate and direct satisfaction, they seek a substitutive satisfaction, as in day dreaming. But the ego impulses have to submit early to the guidance of the reality principle. They are the instincts of self-preservation and have, therefore, to learn to evade real dangers immediately. The guidance of the sexual libido by the reality principle is accomplished only when the ego instincts, which have learned very much earlier to submit to the reality principle, come into relation with the sex instincts. The primary psychic activity (the activity guided by the pleasure principle) is related most closely to the unconscious; the secondary psychic activity (guided by the reality principle) is related to the foreconscious and conscious. Libido, on the one hand, and ego instincts, on the other, are another aspect of this antinomy.

The second point fundamentally changed in this book is a different formulation of the two opposing forces in mental life designated before as the ego and sex instincts.

The speculations contained in "Beyond the Pleasure Principle" took their origin from clinical studies of traumatic neuroses during the war. The attention of Freud was drawn to two facts concerning these traumatic neuroses. The first is the importance of surprise and fright as pathogenic factors. The second is the fact that the occurrence of an organic trauma, i. e., a wound, tends to prevent the development of a neurosis.

In reference to the first of these two factors, Freud distinguishes anxiety, fear and fright. Anxiety is a state of anticipation of danger and of preparation for it, even if the danger is unknown. Fear implies a definite object. Fright is a state characterized by the element of surprise and thus occurs when, without any preparation, one is confronted with a danger situation. In the traumatic neurosis, "the anxiety preparation" is lacking. *Anxiety*, in fact, would prevent the traumatic neurosis; *fright* is what causes it. Freud therefore speaks of fright neurosis. Anxiety, viewed as a preparation against danger, has therefore a special preventive significance against the traumatic neurosis.

Freud explains the preventive function of anxiety by introducing the concept of "defense against stimuli" (Reizschutz). The living organism has not only the function of reception of stimuli, but the almost more important one of defense against stimuli. The organism has to protect itself against being overwhelmed and crushed by the multitudes of impressions continually pouring in from the outside. It has to exclude most of these impressions, and receive only a few which shall serve as samples of the objects in its environment. The anxiety preparation causes a peculiar distribution of libido which prevents a breaking down of the "defense against stimuli" by great excitation. "Such excitations from the outside which are strong enough to break through this defense against stimuli are called traumatic." The traumatic neurosis, therefore, is a consequence of an extensive breaking down of the organism's defense against stimuli.

If circumstances are different, i. e., if an organic wound occurs at the time of the original fright situation, the narcissistic libido investment of the suffering organ creates a similar beneficial equilibrium of distribution of the libido, and this prevents the traumatic neurosis. The anxiety preparation in danger situations occurs on the basis of the pleasure principle or, in other words, of the economic principle.

In a common symptom of the traumatic neurosis, or "fright neurosis," Freud discovered an important exception to the psychanalytic doctrine that dreams are invariably wish fulfilments and always based on the pleasure principle. Anxiety dreams and punishment dreams, according to the psychanalytic doctrine, previously did not constitute such exceptions. Punishment in a dream, for instance, might imply that the pleasurable deed for which the punishment was appropriate was now committed.

Patients suffering from traumatic neuroses, however, often, in dreams, relive and reexperience repeatedly the pathogenic experiences and the fright which attended these experiences. It is hard to see how these dreams could be wish fulfilments. To explain them, Freud introduces a new principle which under certain conditions replaces the pleasure principle. This he calls the tendency to repetition, or the repetition compulsion. He gives three examples of this repetition tendency: First, a child repeatedly hiding an object which it likes and then finding it again. Second, the peculiar constellation in analytic treatment when the patient transfers toward the physician a certain attitude which previously had existed toward another person. The patient cannot give an account of this original constellation, and he is quite unable to put it in its proper place and to recollect it as a fragment of the past. Instead of recalling the original situation, he can only to a certain extent repeat it, using the physician as a substitute for some person of a primary situation. The third example is the fright dreams in which a patient, suffering from a traumatic neurosis, repeatedly reexperiences the original fright situation which provoked the neurosis.

The function of the repetition tendency can be understood in connection with the conception of the defense against stimuli. Before the pleasure principle begins to function, the organism accomplished the psychic binding of traumatic impressions by this more primitive repetition tendency.

The organism's defense against stimuli is directed primarily against the outside world. But the excitations of the deeper mental layers progress directly to the region of the pleasure principle. Certain inner excitations therefore have to be treated as if they came from the outside, in order that the mechanism for defense against stimuli can be used against them. This is the origin of projection.

The second important topic of psychanalysis modified in "Beyond the Pleasure Principle" is the formulation to the effect that part of the ego tendencies are of libidinous nature. The libido can be turned toward objects or toward the ego. We may have then object libido and ego libido. Both tendencies are now included under the term life instincts. The "sexual" libido is that part of the libido which is turned toward objects. On the other hand, the ego itself is the most important object of the libido and that part of the libido which is fixed on the ego, that is to say, the narcissistic libido, corresponds to the instinct of self-preservation or conservation. Freud gives the analogy of a multicellular organism. Each cell lives for itself (ego libido), and each cell tends to a cohesion and cooperation with all the other cells (object libido). The libido turned toward object plus the libido of the ego are therefore the life instincts. And these life instincts are "the Eros of the poets and philosophers."

What, then, is the opposing force to these life instincts? The repetition compulsion shows that there is a tendency in the organism to relive previous states. Freud believes that this is a law inherent in all organic life. As the

organism is assumed to continue to repeat a previous state again and again, it will virtually tend to relive a state before life—that is, death.

Every organism, Freud thinks, has a death tendency. It does not want to die immediately, but it tends toward a manner of death according to its own immanent characteristics. The psychanalytic demonstration of this death tendency can be found in the phenomena of sadism. Freud had believed formerly that sadism, that is to say, the destructive aggressive attitude toward the object, was an original tendency, and that masochism was nothing else but sadism turned against the ego. He now changes this view, and believes that masochism is the original destructive instinct of the ego, corresponding therefore to the death instinct. In sadism, this instinct is transferred to the genital zone. Sadism is a "displaced death instinct." Masochism becomes a regression to a former phase. Instead of the former antithesis between ego interests and libido instincts, Freud arrives now at the contrast between death instincts and life instincts, the life instincts comprising all instincts toward objects or toward the ego that are of libidinous nature.

STAGE OF UNCONSCIOUS PART OF EGO

As belonging to a fifth stage of metapsychology, we may regard Freud's book, "Das Ich und das Es," 1923. He started from the observation that during the analysis resistance is often encountered which comes from the ego itself, the ego being the coherent organization of mental processes in a person. The ego can offer a resistance based on forces which belong to the unconscious. The old formulation of a conflict between the ego and the repressed (= unconscious) is therefore not inclusive enough, since there may be an *unconscious* resistance which belongs to the ego but does not belong to the repressed unconscious.

There are, therefore, three different kinds or regions of the unconscious: (1) The foreconscious is the latent conscious, easily capable of consciousness and therefore descriptively rather than dynamically unconscious; (2) the unconscious in the strict sense, that is to say, the dynamic unconscious repressed; (3) the new contribution of this fifth stage of metapsychology, namely, the unconscious part of the ego. This unconscious part of the ego is called Es, a term introduced by Groddek and previously used by Nietzsche. The Es must be regarded as unconscious for two reasons: first, its forces can be activated without coming into clear consciousness; and second, a great difficulty is encountered when, in the analysis, the attempt is made to lift it to the region of the conscious.

The Es contains the highest and the lowest part of our nature—according to our usual valuations. The highest part is the ego ideal or super ego, which is derived from the Oedipus complex, that is to say, it originates primarly from an identification with the parents. Identification is one of the earliest expressions of "object love."

The Oedipus complex has a four-fold aspect in consequence of the possible ambivalent attitude of the individual toward both father and mother. The loving and therefore feminine attitude of the male individual toward the father is related to the fear of castration by the father, since, according to childhood thought, successful competition with the mother for the love of the father is only possible by giving up the male genital. This fear of castration *by* the

father is often symbolically elaborated in a phantasy of castration *of* the father. In his paper, "A Devil Neurosis in the Seventeenth Century," Imago, 1923, Freud interprets analytically a painting by a neurotic person in which the devil, substituted for the father, is depicted with feminine breasts and these feminine breasts symbolize the castration of the father. The Oedipus complex itself has its origin in the libido, which primarily belongs to the region of the Es.

The individual therefore is conceived in metapsychology as being divided into an Es, which is related to the pleasure principle and is the sphere of instincts and affects, and an ego which is related to the reality principle and is the sphere of perception and reasoning.

In connection with the new formulation of the unconscious, Freud discusses the attempt to abolish the term unconscious and to postulate unnoticed mental phenomena which would be subsumed under consciousness. Apparently Freud refers here to Schilder's conception of a "sphere" (Sphäre), which is thought of as surrounding the clear center of consciousness. This tendency to regard mental processes which the individual does not notice or of which he is not aware as a form of consciousness comes, according to Freud, from the prejudice toward identifying consciousness with the psychic. He gives three arguments as proof of his claim that conscious and unconscious have to be distinguished: First, it needs a decided effort to focus attention on the unnoticed mental content. Second, this unnoticed material, when brought to consciousness, will often not be accepted by the individual with a sense of recognition, but rather it will often be rejected as something alien. Third, Freud claims that if we do not distinguish between conscious and unconscious, we lose the only immediate certainty that exists in mental life. To deny the existence of the unconscious on the basis of the argument that there are only various degrees of consciousness seems to Freud to be analogous to denying the existence of death on the basis of the argument that there are only various degrees of vitality.

Freud evolves a complicated scheme of the topographic relations of the various mental systems. The ego envelops the Es, but only in that part in which the ego itself is in connection with the system of "perceptual consciousness." Ego and Es have no strict delimitation. The system of "perceptual consciousness" receives impression both from within and from without. Sensations from without are either conscious or unconscious. There is no need of assuming a foreconscious for them. Inner thought processes, however, have to pass through the foreconscious in order to become conscious; in the foreconscious they come into connection with word images, and only by doing so can they become conscious. Thus, during the psychanalytic procedure, a repressed content is brought to consciousness through the creation of such foreconscious links. The difference between the foreconscious and the unconscious can therefore be defined not only in their respective relation to the conscious, but by another criterion: that is, their relation to the word images in the foreconscious.

The Es is the great reservoir of the libido. We have, therefore, as a new fact evolved in this fifth stage of metapsychology that when the libido reaches the ego by the mechanism of "identification," we deal with a secondary narcissism. The libido reaching the ego directly from the region of the Es, from which, as we have seen, it is not sharply differentiated, would be primary narcissism.

According to this new scheme, therefore, the ego may be threatened from three sides: first, from the external world; second, from the libido of the Es, and third, from the severity of the ego ideal. Three types of anxiety correspond to these three dangers: first, the object or real anxiety; second, the neurotic libido anxiety; and third, the death anxiety, conscience anxiety and castration fear. In severe cases of neuroses, death anxiety elements are added to neurotic anxiety, which is by far the commonest form. Of these three types of menace or conflict, it is the one between the ego and the ego ideal which can be reached by psychanalysis.

MENINGO-ENCEPHALITIS ASSOCIATED WITH CARDITIS

Report of a Case with Necropsy Findings*

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The association by clinicians of chorea with so-called acute rheumatic fever has had as a basis chiefly the necropsy findings in a few fatal cases. A review of the English literature has revealed that the necropsies were made by those primarily interested in the bacteriologic aspects of the situation or in pathologic investigations that were not thorough so far as the nervous system was concerned. Besides, these investigators did not have at their disposal the present day laboratory facilities and staining methods. In spite of this, Hughlings Jackson,[1] sixty years ago, spoke of chorea associated with fatal cases of acute rheumatic fever as a meningo-encephalitis, and astonishingly enough localized the dominant nervous lesions in the "areas of the brain in and aroud the corpus striatum." H. C. Bastian[2] said that he was convinced from a study of his necropsy material that fatal cases of chorea were types of meningo-encephalitis, and he more definitely even than Jackson placed the principal lesions in the basal ganglia—chiefly the corpus striatum. Many years later, Boynton and Paine[3] reviewed this subject, and reported cases of their own. Their work confirmed that of Hughlings Jackson and Bastian. The following case is of some interest in this connection because it strengthens the conception of chorea as an encephalitis.

REPORT OF CASE

History.—R. J., aged 2 years and 4 months, was admitted to the babies' wards of the New York Post Graduate Hospital on June 1, 1923. His parents had four other children living, none dead. The child was born at full term after a normal birth. He weighed 7½ pounds (3.4 kg.) at birth. He had his first teeth at the age of 9 months; he sat alone at the age of 1 year; he talked

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*Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June, 1924.

1. Jackson, Hughlings: Note on the Embolic Theory of Chorea, Brit. M. J., Dec. 23, 1876; London Hosp. Rep. **1**:459, 1864; Lancet, Nov. 26, 1864, p. 606.

2. Bastian, H. C.: Remarks on the Pathology of Chorea, Brit. M. J. **1**, 1866.

3. Boynton and Paine: Researches on Rheumatism, London J. & A. Churchill, 1913, pp. 239-253.

at the age of 20 months and walked at the age of 21 months. He had had a sore throat several times, chickenpox and otitis media on one occasion. About two weeks prior to admission, a horseshoe thrown by another child struck him on the left side of the head. The next day, the mother noticed that the child walked with difficulty, apparently on account of pain and weakness in the extremities. He was kept in bed for a week or sitting on a chair, because he seemed disinclined to make any attempt to walk. He did not sleep well at night, was restless and complained of pains in the right hand and foot. The joints did not appear red or swollen but were slightly tender. The child's personality changed. Instead of being happy, playful and contented, he became listless, quiet and irritable. He also began to bite his nails. For a week before admission he would not eat. He took only milk.

Examination.—On admission, the patient was found to be a well-nourished, well-developed boy, who did not appear to be acutely ill. There was no sign of injury to the skull or the scalp. The fontanels were closed. The cranial nerves were normal. All the pupillary reactions were normal. Examination of the throat was negative. The postauricular glands were somewhat enlarged. The chest was normal. The heart was not enlarged, and the sounds were of good quality. There were no murmurs. The extremities were normal. There seemed to be no special point of tenderness. The deep reflexes were normal, and the abdominal reflexes were present.

Three days after admission, the rather prominent frontal bosses and beading of the ribs—greater on the left side—were noted. The fundi were normal. The pupillary reactions were normal; no paralysis of the tongue, palate or face was noted. The oculomotor movements were normal during the entire illness, and there were never any pupillary disturbances. All the superficial and deep reflexes were active and equal. There were no pathologic reflexes, no ataxia, and the gait was normal, although the child was distinctly averse to walking. He was mentally keen, responsive and interested when spoken to, but was otherwise listless. A week later, there developed myoclonic twitchings of the abdomen, lids, lips, feet and hands. These movements were very small in their excursion, of very mild force, irregular rhythm and rate and were present during sleep. They were not sufficient to move the entire extremities, but only the small joints and perhaps the foot and hand. The diagnosis made was the choreic type of encephalitis.

On June 13, 1923, the movements involved the neck for the first time, especially when the child sat up, but otherwise continued as before. Thereafter, the condition became acute and of medical interest chiefly because of the development of an endocarditis and bronchopneumonia.

On June 23, 1923, the liver extended three finger-breadths below the costal arch, the heart dilated, the pulse was 160. The heart sounds were poor, and there was a double murmur at the apex. The child died on June 24, 1923. During the child's illness, he ran an irregular temperature, which during the first week did not rise above 101.4 F. The temperature rose chiefly at night. During the second week the temperature continued about the same, rising in the evening to 101-102 F., and in the morning to 99 (all rectal). On June 16, 1924, the temperature rose and stayed up constantly until death when it was about 103 F.

Throat cultures taken at various times during the child's illness were negative, except for various cocci and yeast cells. Urine examinations were essentially negative. A spinal fluid examination was made on June 8 and June 19. The fluid was clear, and the tests for globulin were negative. The spinal

fluid Wassermann test was negative, as was also the colloidal gold test. There were no cells on either occasion. The sugar content of the spinal fluid was 0.085. Repeated blood cultures were negative. The roentgen-ray examination confirmed the heart and lung findings. A roentgenogram of the skull was negative. Necropsy was performed on June 24, 1923, eight hours after death.

THE NECROPSY FINDINGS

The diagnosis at necropsy was acute rheumatic endocarditis, acute encephalitis, fibrinous pericarditis and subacute myocarditis of rheumatic origin. The anatomic findings were fibrinous pericarditis, recent vegetations on the mitral and aortic semilunar valves, an old patch on the tricuspid valve and congestion of the meninges. There was marked enlargement and injection of the lenticulostriate artery on both sides.

The brain grossly showed no marked defects, except a slight turbidity in the pia, with evidences of hyperemia over the entire brain, most marked at the base. Sections were made from various parts of the cerebral cortex, cerebellum, globus pallidus and striate body. These sections were stained with (hematoxylin and eosin) toluidin blue, iron (hematoxylin) and by the van Gieson method.

Microscopic Findings.—Cerebral Cortex: Irregularly over the cortex the meninges were thickened, hyperemic and edematous, and the tissue spaces contained some free blood cells, but, on the whole, little infiltration. The infiltrating cells in the main were lymphocytes. In the cortex itself, there was considerable hyperemia, with formation of new blood vessels. The Virchow-Robin lymph spaces were somewhat dilated and contained a few cells, most of them having the general appearance of lymphocytes, but others were definite plasma cells; nowhere was this cellular infiltration marked, and it was seen defintiely only in the superficial portions of the cortex. This condition did not obtain in the white matter. The nerve cells of the cortex showed little marked change, except a tendency to stain diffusely without clear demarcation of the tigroid bodies. On the whole, the picture was that of a rather moderate stage of acute cloudy swelling. There was no actual cell destruction to be seen. The neuroglia cells of the cortex were definitely increased in number. No neuronophagia was observed. No pathologic forms of neuroglia cells were present. In the white matter of the cortex, the neuroglia cell increase was more marked. In the neighborhood of the blood vessels, phagocytic glia cells containing granules of degeneration products were observed in small numbers.

Basal Ganglia: The pia of the base was somewhat more thickened than that of the cortex. The blood vessels were dilated and filled with blood cells, and appeared to be increased in number. The large vessels which enter from the base were accompanied, in the Virchow-Robin sheaths, by a marked infiltration of lymphocytes. In places, the lymphocytes appeared to have escaped from the vessel sheaths and lay in more or less rounded packed clumps in the tissue close to the vessel. This condition was to be noticed especially in the putamen, close to the external medullary lamina of the pallidus. The large ganglion cells showed little change. The tigroid was present in normal amounts, and the cell shape was not altered. The small ganglion cells showed change in a slight diminution in the amount of tigroid, but this was not striking. There was no cell destruction or cell degeneration. The neuroglia showed a generalized diffuse cellular increase of but moderate extent, with no tendency to the formation of pathologic cell types.

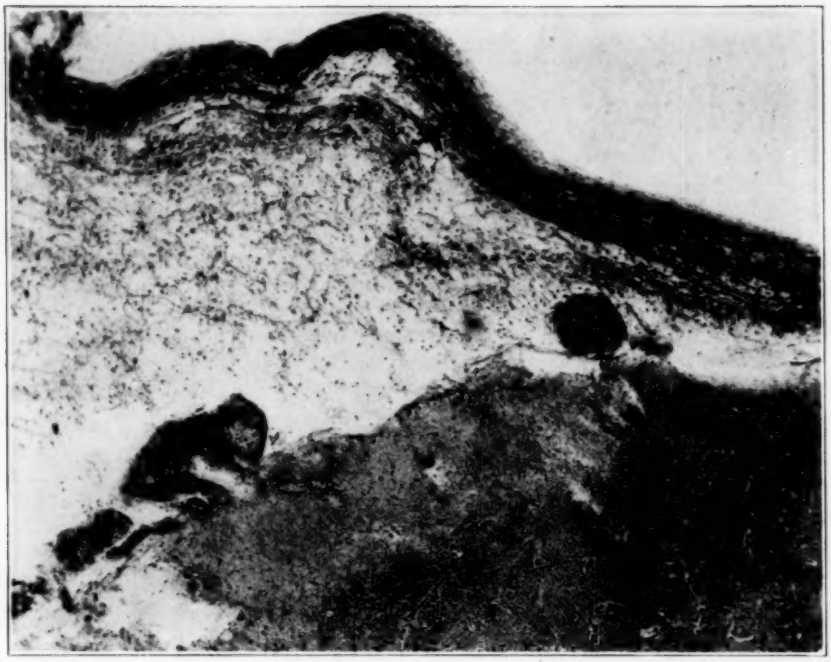

Fig. 1.—Pia over cerebral cortex showing hyperemia, edema and infiltration with lymphocytes.

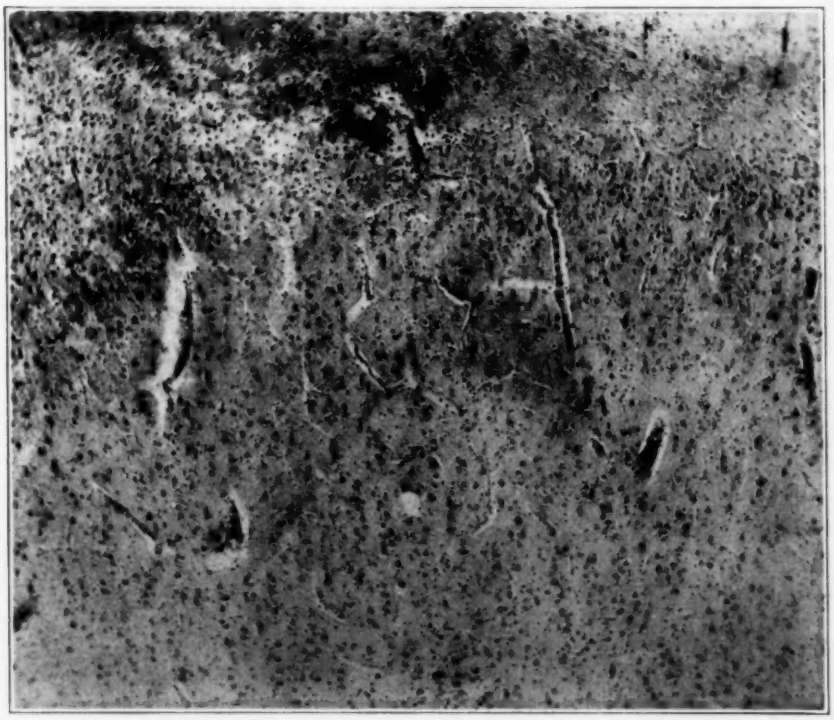

Fig. 2.—Cerebral cortex showing hyperemia, formation of new vessels, absence of infiltration into Virchow-Robin lymph spaces. Increases of cellular neuroglia.

Fig. 3.—Pia at base of brain showing increase of vessels and marked lymphocytic infiltrations.

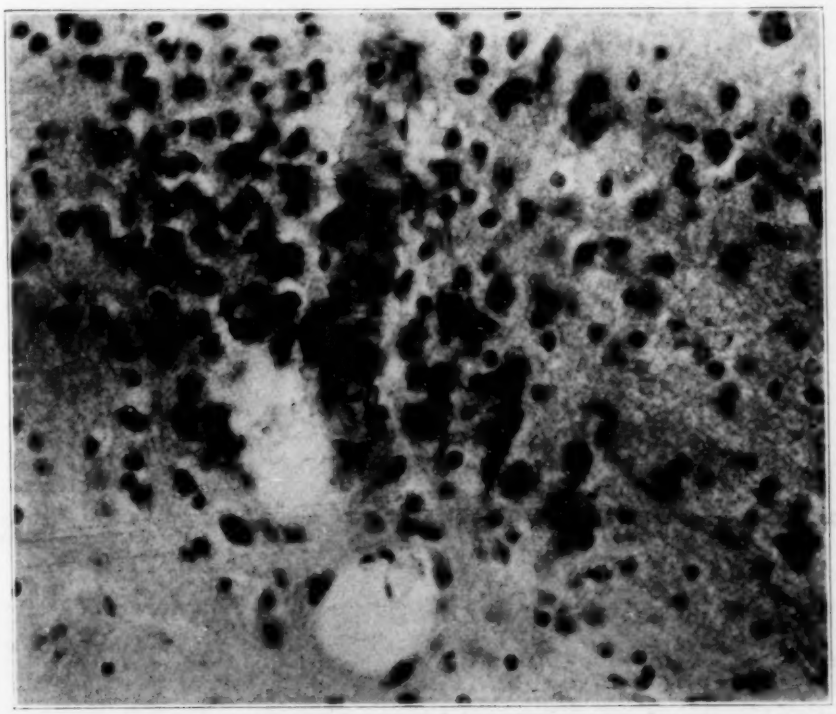

Fig. 4.—Putamen showing free-lying lymphocytes near blood vessel.

Cerebellum: The pia of the cerebellum showed some edema, a slight hyperemia and small scattered patches of infiltration of lymphocytes. The blood vessels of the cerebellar cortex showed no change, and the Purkinje cells appeared to be normal in their numbers and morphology. There was no neuroglia cell increase.

Over the base of the pons, the pia was thickened, hyperemic and infiltrated with lymphocytes. There was some infiltration into the larger vessel sheaths, but this did not extend into the smaller ones. There were no particular changes to be noted in the cells of the pontile nuclei nor in the nuclear or other cells of the tegmentum. The findings in the heart muscle, endocardium, and pericardium, will be made the subject of an extensive report by Drs. Meeker and Osnato, to be published later.

Abstracts from Current Literature

"Hyptokinesis" and "Rubral Ataxia."—A Symptom of Cerebral Tumor of the Middle Cranial Fossa, Especially of the Mid-Brain. The Nucleus Ruber System as an Equilibratory Center. Arthur von Sarbó, Klin. Wchnschr. **1**:1597 (Aug. 5) 1922.

While studying cases of epidemic encephalitis presenting the "amyostatic" syndrome (Strümpell), the author was struck by the large number of patients complaining of symptoms referable to disturbances of equilibrium. These patients had great difficulty in maintaing the erect posture. When attempting to do so, they found that the upper part of the body was continually swaying backward. This was even noticed in patients who had no retropulsion. The phenomenon was designated by Sarbó "hyptokinesis," (*τό ὑπτίασμα* = bending backward). It could be elicited as follows: The patient was ordered to stand erect, with his feet closely together (as in testing for the Romberg sign); the chin was then pushed back, so that the back of the head was bent at an angle with the dorsal surface of the body, when the upper part of the body was found to begin to sway backward. When this procedure did not produce swaying, the patient was ordered to close his eyes. If *this* did not bring out the swaying, the test was considered negative. Sarbó could elicit this phenomenon in almost every case of "amyostasis," and also in every case of paralysis agitans of prolonged duration. He regarded the symptom as an early manifestation of retropulsion, because in cases in which it was marked, the "swaying backward" was followed by retropulsion, "the patient ran backward in order to maintain his equilibrium." There was no doubt that the phenomenon had some disturbance of equilibrium as its basis, although its precise localization in the central nervous system, was unknown. It seemed, however, to bear some relation to disease of the striatum, and when the author discussed "hyptokinesis" for the first time, in connection with epidemic encephalitis (1920), he was of the opinion that the nucleus ruber might possibly be the localization for the phenomenon in question. He was led to this belief because the nucleus ruber, situated, as it is, in the hypothalamus, serves as the connecting center between the striatum and the dentate nucleus, and he was fortified in this belief by the fact that the hypothalamus is found so frequently involved in epidemic encephalitis.

Shortly after Sarbó had published his views on "hyptokinesis" in epidemic encephalitis, he accidentally found the symptom in a patient with undoubted evidences of cerebral neoplasm. The presence of a generalized neurofibromatosis in connection with the neurologic findings pointed to a tumor of the eighth cranial nerve, and suboccipital decompression was advised. The presence of cerebellar gait in addition to the "hyptokinesis" ("hyptokinesis" could not be elicited when the patient was in the sitting posture) was in Sarbó's opinion a deciding factor in the localization of the neoplasm. The patient died soon after the decompression, and necropsy revealed a solid tumor of the size of a hazelnut (a glioma) involving the left mammillary body, a marked internal hydrocephalus, and a normal hypophysis. The tumor had compressed the chiasm and left optic tract. Macroscopically, the cerebellum and the red nuclei were

found to be intact. This case, according to Sarbó, showed that "hyptokinesis" and a cerebellar gait may also occur in lesions of the middle fossa, without involving the cerebellum directly.

Soon thereafter he had occasion to avail himself of the knowledge gained in the preceding case, in making a correct localizing diagnosis in the following case: A 26 year old woman became ill, in November, 1921, with headache, dizziness and vomiting. She had rotatory nystagmus, contraction of the visual field in the right eye, bilateral optic nerve atrophy, varying patellar reflexes and somnolence. With these findings, the patient was referred for decompression, with the diagnosis of "non-localizable" brain tumor. She was examined by Sarbó, for the first time, just prior to operation, when she showed dull mentality, definite *"hyptokinesis"* (swaying backward and to the right), grayish-white optic nerve heads, blindness in the left eye and right-sided temporal hemianopsia. On these findings, Sarbó made the diagnosis of a brain tumor in the middle fossa. The "hyptokinesis" was indicative of involvement of the red nucleus, and the right-sided hemianopsia was attributed to compression of the chiasm; (the presence of hemianopsia on the left side could not be determined on account of the blindness). In order to save the patient's vision and to relieve her other symptoms, a right temporal decompression was advised, and performed. Following the operation, the patient became restless and more somnolent; she developed a left ptosis and jactitation, and died two weeks later. At necropsy, a greenish tumor of the size of a grape was found at the base of the brain in front of the pons, between the peduncles, bound laterally by the temporal lobes. The tumor extended anteriorly as far as the chiasm, which was displaced to the left. The hypothalamus was so compressed that the third ventricle had entirely disappeared. The posterior portion of the growth in its midline compressed both red nuclei, the compression of the latter being visible to the naked eye. The correct localization of this case, in vivo, the author attributes to the presence of "hyptokinesis."

The next case cited by Sarbó occurred in a 33 year old seamstress, with the history that in November, 1921, she struck her head against the edge of a table. There was no loss of consciousness, but she began to suffer from headaches, which became especially intense around the Christmas holidays. At about the same time she developed a memory defect and a lack of apperception. Examination in January, 1922, elicited aphasia, a questionable beginning bilateral papilledema, right homonymous hemianopsia with retention of the hemiopic pupillary reaction, a slight left central facial paralysis, slight ptosis of the right upper extremity and a left Babinski sign. The blood Wassermann test and the roentgen-ray examinations were negative. The patient became progressively worse, and on February 28, there was a definite left-sided and beginning right-sided papilledema. Mercurial inunctions were ineffectual. She also developed a bradycardia. These signs and symptoms led to the diagnosis of a tumor in the left occipital lobe. On the day following admission to the hospital, the patient's relatives said that ever since the accident they had noticed that the patient had evidences of "Witzelsucht," and was confused both in action and speech. She was also forgetful. The patient herself had localized her headache in the left frontal region, which was also tender to percussion. At that time there was no cranial nerve involvement; definite bradycardia and unusual pallor were present. On standing erect with her eyes closed, she was swaying backward. From these facts, Sarbó was led to believe that she had either a traumatic abscess or a tumor in the left frontal region. The right-sided homonymous hemianopsia he regarded as a remote localizing symptom

due to compression of the left optic tract, but owing to the presence of "hyptokinesis," he felt that the midbrain must be involved in the pathologic process. He therefore advised decompression on the left side. The patient died on the same day before any operative procedure could be undertaken. The necropsy revealed the presence of a reddish colored tumor of the size of a nut, infiltrating the right frontal lobe and extending into the wall of the right lateral ventricle and into the corpus callosum; the splenium was markedly increased in size. The tumor had also invaded the columnae fornicis and the septum pellucidum, the cavity of the latter having entirely disappeared. The anterior portion of the third ventricle was also included in the growth, which extended laterally into the mesial portion of the right temporal lobe. Both optic tracts, especially the left, and the left side of the sella turcica had been flattened out by the pressure exerted on it by the tumor. Both red nuclei could be seen macroscopically, diminished in size and changed in form. Sarbó points out that his localization in the frontal lobe was correct, although he made an error in localizing it on the left side. He also states that he was apparently correct in considering the homonymous hemianopsia a tract symptom, and that he was largely aided in this by the presence of "hyptokinesis."

This symptom, the author states, enabled him to localize the pathologic process definitely in the midbrain in both cases. He concludes, therefore, that "hyptokinesis" is a positive sign of involvement of the midbrain, when in addition to it there also exist: (1) bitemporal or homonymous hemianopsia as evidence of optic chiasm or optic tract involvement; (2) evidences of striatal involvement (Strümpell's amyostasis); (3) symptoms of thalamic involvement (thalamic syndrome of Déjerine, Roussy and Head); (4) ptosis, Argyll Robertson pupils (evidences of involvement of the corpora quadrigemina); (5) central diminution of audition (posterior corpora quadrigemina), and (6) pineal symptoms. In addition, every case must also show evidences of hydrocephalus with distention of the third ventricle.

The foregoing cases and a review of the literature with an analysis of the histories of many of the cases, Sarbó states, confirm his views that "hyptokinesis" is due to involvement of the red nucleus in disease of the frontal and temporal lobes, as well as of the corpus callosum and pons. In all these localizations, "hyptokinesis" when present indicates a disturbance of equilibrium, which must be attributed to involvement of the nucleus ruber system, because it has long ago been demonstrated that the proper maintenance of equilibrium is largely dependent on the integrity of this system. The red nuclei are nothing more than outposts of the cerebellum, with which they are connected by the brachii conjunctivae. Furthermore, these nuclei are connected with the striatum, the thalamus, and through the latter with the temporal and frontal lobes. The rubrospinal tract (Monakow) enables the red nuclei to exert their influence on the ganglion cells of the anterior horns of the spinal cord. In this connection, it must also be borne in mind that most of the fibers of Monakow's bundle are broken up in the cervical and lumbar enlargements of the cord. Finally, these nuclei are, in all probability, connected with the column of Goll through the median lemniscus. It is through the latter two that impressions of deep sensibility from the lower extremities are conveyed to the higher centers. Total destruction of the red nuclei should theoretically lead to severe equilibratory disturbances, and this is actually found to be the case. "Hyptokinesis," therefore, Sarbó considers a dysfunction of the nucleus ruber system, whereas total destruction of the ganglion cells of these nuclei (large motor type ganglion cells) leads to rubral equilibratory disturbances, which corresponds clinically

to cerebellar ataxia. Why "hyptokinesis" should consist merely of a swaying backward, can only be surmised. The impression gained by the author thus far is that pathologic processes which involve the frontal connection of the nucleus ruber system give rise to "hyptokinesis," whereas processes caudad to the red nuclei produce a swaying forward. In any given case, this symptom must be taken into consideration with all other symptoms and signs before proper clinical evaluation can be attached to it. Early "hyptokinesis" followed by cerebellar ataxia indicates that the pathologic process is localized in the midbrain.

The association of "hyptokinesis" with striatal symptoms in epidemic encephalitis is striking. This, according to Sarbó, would also seem to indicate that the striatum plays an important rôle in equilibrium. This conception is, to a great extent, confirmed by comparative anatomy. In animals in which unusual demands are made on the proper maintenance of equilibrium, namely, in birds, the striatum is enormously developed. In these animals, the size of the striatum and cerebellum is out of proportion to that of the pallium. All these facts would lead, therefore, to the conclusion that the chief function of the nucleus ruber system is to maintain static equilibrium.

Sarbó's final conclusions are summarized as follows: (1) In almost every case of epidemic encephalitis that presents Strümpell's amyostatis syndrome, this symptom as well as the other symptoms of amyostasis show daily wide variations in their intensity. They may be marked on some days and absent on others; they may also disappear temporarily for days or weeks. Usually, however, there exists a certain parallelism between the intensity of the "hyptokinesis" and that of the other symptoms. This variation in intensity is most probably due to the extent (acuity) of the chronic inflammatory process and to the circulatory disturbances (edema) of the structures adjacent to the centers involved, especially in the hypothalamus. (2) In endarteritic circulatory disturbances following striatal softening. (3) In paralysis agitans. (Before testing for "hyptokinesis" in this disease, it is essential that both the trunk and flexed knees are held in extension.) (4) In tumors of the midbrain (tumors of the corpora mammillaria, pineal body, anterior and posterior corpora quadrigemina, thalamus, and in tumors surrounding the third ventricle). (5) In tumors which exert pressure on the midbrain (frontal, callossal, and temporal tumors). Sarbó believes that callosal ataxia, designated by Bruns as "frontal ataxia," is nothing more than a disturbance of equilibrium due to dysfunction or complete loss of function of the nucleus ruber system. And, as the function of this system may be seriously interfered with by the presence of tumors in the regions named, Sarbó would prefer to designate this form of ataxia as "rubral ataxia" rather than as "fronto-callosal ataxia." (6) In cases of hydrocephalus with obstruction of the third ventricle.

The article is well illustrated.

KESCHNER, New York.

THE INFLUENCE OF TERTIAN MALARIA ON PROGRESSIVE (GENERAL) PARALYSIS. H. REESE and K. PETER, Med. Klin. **20,** No. 12 (March 27 and April 3) 1924.

The following abstract may be regarded as the official pronouncement of Nonne's clinic on this very important topic.

Following Wagner von Jauregg's communication on this subject, the authors began (June, 1920) to apply the malaria treatment to 270 paretic patients in Nonne's clinic. Before detailing the treatment and its results, the authors emphasize that the absolute diagnosis of general paralysis be made with

serologic aid to exclude from consideration other syphilitic conditions. Secondly, they draw attention to the well-known fact that spontaneous remissions in this disease do occur.

Because of the syphilitic aortitis present in so many late cases of paralysis (81 per cent.), the late, weak and decrepit paretic patients were excluded. The report is concerned with the clinical and serologic studies made on these cases over a period of from one to two years following the malaria inoculations. First, the relatives of the patient were asked to give a written permission before the procedure was undertaken. Second, quinin was given to the patient before the injection in order to determine whether there was a quinin idiosyncrasy present. If so, the treatment was not begun. If the malarial blood is not immediately injected, it must be kept warm, 37 C.; if it be transported any distance, it must not be shaken up, lest the plasmodia be jarred out of the erythrocytes and perish. By preference, direct intravenous injection is made, about 2 c.c. or more of infected blood being used. There are no reactions. In from five to eight days, the characteristic chills begin, although plasmodia may be found in the blood a considerable time before the onset of chills. In the course of many injections, there was no definite lengthening or shortening of the incubation period. In 5 per cent. of the patients, no febrile attacks occurred after repeated injections. Some showed neither parasites in the blood nor febrile attacks. Others developed malaria only after the second or third inoculation. Typical febrile curves and blood pictures are presented. No weakening of the organism's virulence could be demonstrated. In some cases, there seemed to be an increase. The patient was not allowed to have more than twelve chills. The occasional presence of a moderate or severe icterus prompted an immediate cessation of the treatment, as does also an agonizing exhaustion of cardiac efficiency even with the support of stimulants. During the treatment one occasionally sees acute mental deterioration. More often motor restlessness and the appearance of paranoid ideas occur, particularly in those not previously manifesting these symptoms. Eighteen developed fatal complications undergoing the malaria treatment. In these, the authors were able to maintain that the lethal issue bore no relationship to the artificially induced infection.

They sound a warning to the effect that the giver of the malarial blood should be carefully examined to exclude the malignant estivo-autumnal form of parasite. Fatal cases have been reported as being due to infection by this organism.

The treatment of the malaria was according to Mühlens' scheme; 0.5 gm. of quinin hydrochlorid was given by mouth in the morning, and again at night for one week. Then there were six or seven quinin-free days. Then alternate periods of three days of 1 gm. quinin a day and six or seven day quinin-free intervals until 15 to 20 gm. had been taken. If immediate cessation were desired, intravenous (or intramuscular) injections of quinin urethane were used. The patients responded well and remained free from recurrences. Indeed, prompt cures with no recurrences were attained with Mühlens' scheme, using only 0.5 gm. of quinin a day. During the period of psychic convalescence, the authors gave a six to eight week course of mercury inunction (4 gm. of mercury a day). They avoided the use of arsphenamin, remarking on its unfavorable influence on paralysis. The complications which made cessation of treatment necessary were marasmus, hemorrhagic cystopyelitis, pachymeningitis hemorrhagica, cardiac weakness, sepsis, etc. They refer to Mühlens'

admonition that quinin be given immediately, preferably quinin urethane, 1 gm., intramuscularly, if there arise a sudden great increase in the plasmodia in the blood stream.

They give statistics on seventy-five cases of their series which have been followed over a period of from one and one-half to two years; and have been serologically and clinically controlled.

Group I, thirty-eight (50 per cent.) show definite unmistakable remissions. By remission, they refer to a clinical improvement associated with an ability to carry on the profession or work. Certain psychic and somatic residua remain.

Group II, fifteen (20 per cent.) show such definite improvement that they are regarded as practically cured. They are back at work, regarded by their fellows as healthy. The clinical examination revealed only the minutest somatic and psychic anomalies.

Group III, twenty-two (29.4 per cent.) were uninfluenced by malaria treatment. Among these were three juvenile patients with paralysis. Why these cases did not respond is unknown. Future anatomic studies may throw light on these recalcitrant cases. Yet a disease so variable as paralysis in its pathologic progress the authors state cannot be expected to respond to *one* method of treatment always in an adequate manner. Perhaps the progress of parenchymal degeneration allowed no restitution, or other intact brain areas were unable to compensate for the parts degenerated and rendered functionless.

Group IV, twenty-five (10.6 per cent.) fatal cases of the entire series; of these, eighteen were unrelated to malaria but to other intercurrent and complicating conditions. The seven remaining (2.9 per cent.) on postmortem examination revealed no particular added cause of death. These were young, strong, apparently robust men, whose death the authors regard as having been due to, or much influenced by, the malaria. One of them had a persistent thymus. Although this percentage is small, it becomes necessary to discuss it with the relatives and to obtain from them their written consent.

In seventy (90 per cent.) of the cases remission occurred. The best remissions were noted in the agitated-manic types. The authors believe that the demented forms offer the worst prognosis. The average age was: Group I, 46; II, 38.3; III, 46.4; IV, 40.8. These remissions resemble in every way those occurring spontaneously. Hoppe gives the spontaneously occurring remissions in the following percentages—for men, 16.8 per cent.; women, 14.9 per cent.; Gaupp, 10 per cent.; Kirschbaum, 5.6 per cent. and 11.4 per cent.

The time of the onset of the remission varies much; many begin soon after cessation of treatment, others weeks to months after. The duration has been from one and one-half to two years. After a six months' period, the authors have repeated their malaria inoculation in certain cases to fortify and intensify the remission (particularly in the psychic sphere).

The authors comment on the serology in the blood and cerebrospinal fluid. They emphasize one fact, that one cannot make prognostic deductions from such examinations. There is no parallelism between psychic improvement and the spinal fluid findings. However, a tabulation of thirteen cases is shown in which are detailed noteworthy changes in the fluid content before and after malaria treatment. In nineteen cases, a definite influence on the albumin and cell count was noted: in seven cases, the blood Wassermann test, and in five the spinal fluid test, became negative. A weakening of the blood Wassermann test appeared in four cases, and in the cerebrospinal fluid in one. A second table shows a dropping of the mastic curve. Of special interest are three

instances which show completely negative spinal fluid reactions and negative blood and fluid Wassermann reactions after the course of several years. From this significant fact, one may infer that a disappearance of serologic reactions can occur in the course of years in patients having a remission. On the other hand, a number of cases occur in which the spinal fluids show no change, despite good, even excellent, remissions. Psychiatrically the improvements are astounding: complete insight, restoration of critical judgment, complete ability to work have returned. Neurologically, the authors' experience warrants the statement that all symptoms of paralysis may be improved by the malaria treatment. They often observed an increase in the reaction of the pupil to light; although it rarely returned to normal. In eight instances, the Argyll Robertson phenomenon disappeared. It is astonishing how often severe speech and writing disturbances are ameliorated. Paralytic attacks subside with recession of pyramidal tract symptoms. A general improvement to a state of well-being occurs which belies the presence of the grave disease. In paralysis the hemolysin reaction evidences an increased permeability of the meningeal blood vessels for amboceptor. The latter hemolyze the sheep's red blood cells and are present in normal human serum. If one treats the sheep's red blood cells with paretic cerebrospinal fluid, the addition of complement produces hemolysis, whereas in acute meningeal inflammations the addition of complement is not necessary. With Weil and Kafka, they have experienced the following with regard to prognosis: the presence of continued hemolysin reaction in the cerebrospinal fluid points to an unfavorable outcome; the absence of it points to a disappearance of all progression and the transition to a stationary type of paresis.

How does malaria produce this beneficial result in this encephalic spirochetosis? The question resolves itself into several parts. Is the fever responsible? Is the effect to be ascribed to a nonspecific general protein therapy which leads to an enhancement of the defensive bodies? Does the malaria lead to the formation of more or less specific antibodies against the spirochete or its toxin?

One must admit that there is no specific reaction, otherwise the various infectious diseases and hyperpyrexias would not effect the same favorable response. Fever alone does not produce the remission. Excellent remissions were noted in cases associated with only a slight rise in temperature. Plant and Steiner, working with recurrent fever, believe that the immunity-bodies of this disease are active against *Spirochaeta pallida*.

Nor is a leukocytosis responsible, for it does not appear following the malaria injection. Jahnel found extraordinarily active spirochetes in a person with general paralysis who suffered from a purulent meningitis. He concluded that "if such a strongly purulent process in the brain has no effect then no artificially induced leucocytosis can be of any avail." All theoretical explanations concerning the effect of malaria on general paralysis are unsatisfactory. The authors agree with the results of the researches of E. F. Mueller. He believes that following various injections, especially intracutaneous, changes in vascular tonus occur in various parts of the body. According to Mueller, parasympathetic effects, such as vasodilatation, take place, leading to local hyperemia and transudation. These local reactions are accompanied by polymorphonuclear leukocytic invasions and escape of serum. Such a leukocytic process occurs without a general hemic leukocytosis and is conditioned by a parasympathetic vasodilatation. The general leukocyte count in the peripheral

circulation and the blood pressure falls. This is just what happens at the onset in the chill in malaria. In fact, the general leukopenia in the peripheral blood stream indicates a shunting of the current and cells into the abdominal blood vessels, which are dilated by parasympathetic impulses; while the abdominal vasodilatation is quickly neutralized by the intact antagonistic vasoconstrictors, the dilatation occurring in these local inflammatory-like areas is of longer standing and leads to the focal changes above described. This conception, then, postulates an effect on the vegetative vasomotor system independent of temperature rises, yet conditioned by malaria. Our aim does not stop here. We must attempt to impregnate these locally occurring transudate-processes with our specific therapy (mercury-arsphenamin, etc.) so as to bring our antisyphilitic remedies into the most intimate contact with the spirochetes.

The authors conclude that this malarial therapy is the method of choice in general paralysis.

BROCK, New York.

THE EMOTIVE ACTION OF EPINEPHRIN. G. MARAÑÓN, Libro en honor de S. Ramón y Cajal **2**:291-310, 1922.

Marañón reports the results of experiments of injections of epinephrin in doses of 0.5 to 1 mg. of a 1:1,000 solution, which produce emotional effects, already described by him in 1920. The experiments were carried out during two years on 130 human subjects, some healthy and others suffering from different diseases. After reviewing the effects produced by injections of epinephrin into the human organism, the emotional reaction is described in detail.

The emotional phenomena, usually manifested shortly after the injection (about fifteen minutes or more) appear under two different aspects. In some cases, they are manifested as a simple subjective perception of certain somatic disturbances which spontaneously arouse in the patient an indefinite emotional sensation, but "coldly" perceived, without emotion proper. The subject is conscious of the phenomena following the injection of epinephrin, such as internal tremor, tremor of the limbs, precordial pressure, chills in the back, cold hands, parched mouth, palpitations, shedding of tears, and, as these phenomena coincide with the complete picture of the vegetative symptoms of emotion, their perception arouses the sensation of being in an emotional condition, without the presence of the psychic element of emotion. The patients, either spontaneously or after being asked, describe their condition with phrases such as the following: "I feel as if I were afraid," "as if I were expecting a great joy," "as if I were going to weep without knowing why," "as if I were in great fear, yet I am calm," "as if somebody were going to hurt me," etc.

In the second group of cases, less frequent, the patient not only perceives the somatic vegetative modifications characteristic of emotion, but he also feels, gradually or suddenly, psychic emotion, the result being a complete emotional outburst usually accompanied by weeping, sobbing and sighing. Usually a psychic motive of sad nature appears and is superimposed on the emotion, the patient thinking of absent or dead relatives or of some other painful details of his life. But in some instances the psychic motive does not appear, and the patient acknowledges that he is weeping without knowing why. This second degree of epinephrin emotion appears spontaneously in some cases, while in others it must be elicited. Some patients were asked before the injection details about their absent or dead relatives, without

arousing in them any sad feeling; the same questions put to them after the epinephrin injection were enough to bring about an outburst of sobs and weeping.

The emotional reaction does not appear in all the patients; of 130 subjects, the reaction was positive in thirty-one (23 per cent.). Most of the positive reactions were observed in patients suffering from hyperthyroidism. Marañón thinks that the condition of emotional instability characteristic of these patients constitutes an important predisposing circumstance. Evidence favoring this interpretation is found in the fact that a negative reaction can be changed into positive when the patient is previously treated with a thyroid preparation. Two examples of this inversion of reaction are described in detail. Conversely, hypothyroidism either hampers or suppresses the reaction. In eight cases of myxedema, the reaction was negative even though among these patients there were two who suffered from a habitual emotional condition. There are, however, many cases of typical hyperthyroidism in which the reaction is negative, and there are also patients suffering from hypothyroidism who show a positive reaction. On account of these facts, the reaction has no diagnostic value.

The reaction is often positive in those psychopathic transitory conditions accompanied by deep emotional instability usually occurring in women during the menopause. In some of these women, there was no hyperthyroidism, as shown by a careful clinical examination and by the determination of basal metabolism, which was negative. It seems, therefore, that the early appearance of the reaction is not always linked with the thyroid factor, but with some other pathologic conditions. That one of these conditions is not brought about by ovarian insufficiency, is proved by the fact that in seven patients suffering from juvenile ovarian insufficiency the reaction was always negative, although in two of them, one with hyperthyroid reaction, the other suffering from a clearly hysterical condition, a positive reaction should be expected *a priori*.

In those conditions of climacteric emotional instability, there is usually a parallel instability of arterial tension with tendency to hypertension. Many authors, with whom Marañón agrees, suppose that this circulatory condition might well depend on suprarenal hyperfunction; in such persons, epinephrin should produce more effect than in subjects with normal or hyponormal suprarenal activity. That such is not the case, is proved by the fact that injections in four cases of Addison's disease produced an intense emotional reaction. This result coincides with the frequency of a positive reaction in patients recovering from severe infectious diseases, in whom suprarenal activity has perhaps decreased during the struggle against the infection. A positive reaction is also obtained in neurotic patients of the emotional type who do not show clinical symptoms of endocrine disturbances, and this result is in agreement with the results reported by Sierra in patients suffering from melancholia, depressive mania and dementia praecox of the catatonic type.

In normal persons, the reaction is always negative, but may become positive in cases of emotional excitation. Marañón has observed a positive reaction in patients with surgical lesions in whom emotional outbursts are produced after the injection, owing to fear aroused in the subject by the approaching operation. He also mentions as further proof the experience of dentists who use procain or cocain associated with epinephrin as an anesthetic; some of the patients, apparently excited by pain and insomnia, and also through fear of the operation, may suffer from symptoms such as tachycardia, paleness,

goose flesh, tremor, restlessness, weeping, etc., which usually disappear within a short time. These symptoms, he believes, are nothing but emotional reactions following the injection of epinephrin. From his observations, the author concludes that the emotion following injection of epinephrin usually appears in subjects with a high emotional index, and that the latter seems to be linked in a great number of cases with hyperthyroidism; furthermore, it can be artificially produced by a previous treatment with thyroidin in sufficient quantity.

There is not absolute parallelism between the emotional and the local and general reaction produced by the epinephrin. In some cases, the emotional reaction is intense, while the local and general reaction is not so manifest, and vice-versa. The same dissociation is found between local and general phenomena. In general, it may be said that intense emotional reactions are found in patients in whom arterial pressure was increased from 10 to 20 or more millimeters, while less intense and negative reactions occur in subjects with invariable pressure or with hypotension.

The mechanism of the emotional phenomena produced by the injection of epinephrin is discussed. Marañón believes that epinephrin determines a diffuse excitation of the vegetative nervous system in its sympathetic portion. The visceral phenomena, being to a large extent the same that occur in spontaneous emotions, appear to the individual as an emotion which is "coldly" perceived (first degree of reaction) or is felt as a complete or authentic emotion (second degree of reaction). The identity of emotional phenomena after injection of epinephrin with the "emotional reflexes" of psychologists is emphasized by the following comparison:

Emotional Reflexes (according to Achille-Delmas and Boll)	*Visceral Phenomena After Injection of Epinephrin*
Screaming and moaning, sobbing	
Sighing	Sighing
Weeping	Weeping
	Polypnea
Tremor	Tremor
Chills	Chills
Easily startled	Easily startled
Nausea	Nausea
Esophagic and pharyngeal spasms	Visceral spasms
Intestinal and vesical spasms	Vesical spasms
Shedding of tears	Shedding of tears
Salivation	Salivation
Perspiration	Perspiration
Polyuria	Polyuria
Diarrhea	
Blushing	Blushing
Paleness	Paleness
Palpitations	Palpitations
	Tachycardia
	Hypertension
	Hyperglycemia
	Glycosuria

The dissociation between the vegetative and psychic elements of experimentally induced emotion by the subject of the experiment is of great importance for the understanding of the problem of the emotional mechanism. The "cold" perception and consciousness of the vegetative phenomena of emotion occurring in the first degree of the reaction deals a heavy blow to the opinions of James

and Lange, who claim that psychic emotion is the result of the perception by the brain of these peripheral vegetative phenomena characteristic of emotion. In the second degree of the reaction, psychic emotion is secondary to vegetative emotion, but this happens less frequently; this mechanism must, therefore, be regarded as exceptional, instead of being the normal mechanism, as claimed by James and Lange. According to Marañón, the physiologic process of emotion can be expressed in a diagrammatic way as follows: (1) initial psychic element (sensation, idea, recollection); (2) production of peripheral or vegetative emotion; (3) consciousness of this peripheral emotion by the brain; and (4) authentic emotion when this consciousness of vegetative phenomena is superimposed on the primitive psychic element.

With regard to the physiologic meaning of the emotional reaction, Marañón states that the specific effect of epinephrin cannot be regarded as purely pharmacologic results, and suggests that a sudden release of epinephrin in the organism might well contribute toward the production of the diffuse and varied peripheral symptoms of emotion. He concludes that among the drugs used in pharmacology or in physiology there is none possessing the emotive power of epinephrin.

NONIDEZ, New York.

ON CEREBELLAR LOCALIZATION. SVEN INGVAR, Brain **46**:301 (Oct.) 1923.

"Our knowledge of the cerebellum culminates in a few very important facts, all of which indicate that a great fundamental and uniform function is represented in this organ." These fundamental facts may be stated as follows:

1. The cerebellar cortex shows no differences of histologic structure. Not only have all animals the same layers of cells in the cerebellar cortex, but the structure is the same in all parts of the cortex from the simplest cerebellum to the most intricate—that of man.

2. The cerebellum is built up in the same manner in all animals. This has been demonstrated in mammals by Bolk.

3. In all vertebrates without exception the cerebellum develops in the same part of the neuraxis.

"Naturally, all these facts indicate a uniform function in all animals, and a uniform function in all parts of the cerebellar cortex, but they do not exclude a localization of function. . . . There is a strong argument for localization in the distribution of the afferent fibers to the organ, since the different afferent tracts are distributed to different cortical regions. Such an arrangement must undoubtedly mean that in different regions different functions are localized."

Bolk made the first important contribution to our knowledge of cerebellar function. He divided the cerebellum into two parts, the lobus anterior and lobus posterior, separated by a deep fissure, the sulcus primarius. Ingvar, however, divided the cerebellum into three main parts, a lobus anterior, a lobus posterior and between them a lobus medius. This subdivision of Ingvar, based on comparative studies, has been accepted by Kappers, Ransom, Bremer and Marburg.

Concerning the function of the middle lobe of the cerebellum, there is a pretty general uniformity. The middle lobe is made up of the lobuli ansiformis and paramedianus, these appearing first in mammals contemporaneous with the pons and the pyramidal tracts. The middle lobe receives most of the pontile fibers to the cerebellum. Bolk concluded from his anatomic studies that the lobi ansiformis and paramedianus must be functionally related to the activities of the extremities. He inferred that the anterior part of the anterior lobe is

related to the anterior extremity, while the posterior region has the same relation to the hind limb. His anatomic studies were succeeded by a long series of experimental observations by van Rhyneberk, Luna, Rossi, Lussana et al., all of which have confirmed his views of the arrangement of the centers for the anterior and posterior limbs in animals. Hesser and Troell were unable, however, to corroborate this localization. Rossi, after an exhaustive study, concluded that the fore limb is related mainly but not exclusively to the anterior crus, and the hind limb mainly to the posterior crus and the paramedian lobe. "Altogether almost all authors agree that the lateral parts of the lobus medius are in functional relation with the extremities. So far the question of localization in the cerebellum seems to be settled."

There is no such uniformity of agreement about the anterior and posterior lobes, however. The author believes that these parts have a fundamental function. His views in this regard are based on certain anatomic facts which may be stated briefly as follows: (1) The anterior and posterior lobes of the cerebellum have changed little during phylogenetic development. (2) These lobes receive the spinocerebellar fibers which originate from all segments of the spinal cord but which are not segmentally represented in the cerebellar cortex, making it probable that these regions have a function correlated to all spinal segments—i. e., the body or organism as a whole. (3) The fact that the anterior and posterior lobes receive fibers belonging to the vestibular nerves points in the same direction.

That the anterior and posterior lobes do have a fundamental function and are intimately related to the vestibular nerves has been demonstrated in many ways. Ingvar showed experimentally that to these lobes direct fibers go from the vestibular ganglia. Greggio supported this experimentally by demonstrating a defective development of the cerebellum, particularly of its most posterior parts, by excision of the peripheral vestibular organs in new-born pigeons. Mesdag, by ontogenetic studies, showed how vestibular fibers run only to the most posterior parts of the cerebellum. Moreover, the tendency for irritation of a vertical or a horizontal canal to produce nystagmus, past-pointing and falling in the same plane forces the author to conclude that "there must therefore be a grouping of vestibular impulses in the central nervous system," and that this grouping takes place in the cerebellum and is arranged on the principle of the different planes of space. Ferrier and Hughlings Jackson held similar views. Experimental evidence, as well as clinical, also seems to support a grouping of vestibular function in the cerebellum. For example, lesions of those parts of the cerebellar cortex innervated directly from the vestibular nerve produce in the acute stage the so-called forced movements which in many respects remind one of pure vestibular symptoms. Physiologic experiments show that after lesions of the lateral parts of the cerebellum "forced movements" occur, the animal rotating around its own axis and falling to the side of the lesion. Forced movements in the saggital plane occur when the vermis is injured or destroyed. Ingvar has demonstrated experimentally also that when the posterior lobe of the cerebellum is totally removed the animals show a remarkable tendency to fall backward. Destruction of the anterior lobe produces different phenomena, and he therefore concluded that "the synergies necessary for maintaining the body balance backwards were regulated in this posterior part of the cerebellum." Other authors have found a tendency to fall backward, however, after destruction of the anterior lobe. Russell confirmed Ingvar's findings. Simonelli too, has demonstrated the tendency to fall backward on removal of the posterior lobe. He found such a tendency only after

ablation of the most posterior parts of the cerebellum, the uvula and nodulus. This agrees with the work of Ingvar who concludes that "the fact that the falling backwards is elicited especially from those parts innervated by the vestibular nerve makes it very probable that the vestibular innervation plays some rôle in this phenomenon," and that this assumption is all the more probable since this falling backward is almost similar to that which appears after lesions of the vertical semicircular canal.

Ingvar, in 1918, put forth the theory that the cerebellum regulates muscle tone in the different regions of the body in all muscular activities. He decided that there was no regional representation of the body segments within the cerebellar cortex of the anterior and posterior lobes, but that what is localized there is the complex of different synergies acting in equilibrium, according to the principle of the direction in the different spacial planes. "In these parts the regulation of the synergies necessary for the static and dynamic equilibrium of the body as a whole is laid down." Bolk concluded from his studies on animals that the anterior lobe is related to certain definite segments, and so also the posterior lobe. Ingvar, however, feels that the functions of these parts are related to the body as a whole and not to any special segment of it. Gordon Holmes tends to accept Ingvar's conception. Stated briefly, Ingvar's idea is that the cerebellum serves as a regulating organ of muscle tone in order to preserve and maintain the static and dynamic equilibrium of the body masses.

There exists a complete homology between all parts of all cerebellums except in the case of the bony fishes. Therefore the same system of functional localization must hold in all.

Clinical observations seem to corroborate in a general way the principles evolved above. Nothnagel states that a lesion of the vermis causes falling in a forward or backward direction. Adler, Brum, Stewart and Holmes have reported cases, but there is no general agreement about the direction of the falling in a lesion of the vermis. It is generally felt, however, that when the lateral parts of the cerebellum are affected the patient falls sideways, while a lesion of the vermis causes a falling in a more or less sagittal direction. Thomas reports the case of a man in whom the symptoms were limited to the right arm. Postmortem examination showed an abscess in the right superior semilunar lobe. Rothmann reports a case in which he was able to diagnose a cerebellar abscess in the posterior part of the hemisphere owing to the fact that ataxia had begun in the leg. Leri reported a case of a wounded soldier who developed cerebellar symptoms in the legs only, after a lesion of the inferior vermis. He decided there was in man in the inferior vermis a center in charge of the coordination of the motility of the legs. The site of the lesion, however, was not verified, and so the observation loses in importance. Gordon Holmes is not in favor of a strict localization or of a circumscribed functional representation of different parts of the body in the lateral lobes.

The author reports two cases which illustrate the localization in man. One case was that of a man with tumor in the right lobulus biventer, whose only clinical sign was cerebellar ataxia of the right leg. The other case was a general ataxia following extensive injury to the right hemisphere after operation. The right arm was chiefly affected, the severe general ataxia disappearing after a time and leaving the arm and leg only affected. Necropsy showed the lesion to be in the right hemisphere.

ALPERS, Philadelphia.

PHYSIOLOGY AND PATHOLOGY OF SKELETAL MUSCLE TONUS. E. A. SPIEGEL, Ztschr. f. d. ges. Neurol. u. Psychiat. **81**:517-624, 1923.

The author gives a lengthy and complete summary of his subject, with a review of most of the work done up to this time and results of his own experiments.

Tonus means muscle tension and is due to centripetal impulses and the resultant reflex actions. Muscles not only have elasticity but also plasticity in maintaining any degree of tension. This was first shown by Sherrington in decerebrated cats. Muscles, therefore, are used both as a means of movement and as brakes on their antagonists. Kinnier Wilson in his description of progressive lenticular degeneration showed that motor systems quite different from the pyramidal tracts were involved. Struempel characterized all such disturbances as amyostatic syndromes.

Accessory nerve end-plates consisting of sheathless fibers were first demonstrated by Boecke. Taking these findings into consideration, De Boers tried to prove that muscle tonus was due to nerve impulses derived from the thoracic autonomic nervous system, but this was disproved by Cobb, van Rijnbeck, Dussen de Barenne and others.

Siegel was unable to show a tonus difference of the two anterior extremities of male frogs, showing a prehensile reflex during the mating season after the thoracic sympathetic ganglia of one side had been removed.

The theory that parasympathetic innervation of the skeletal muscles produces their tonus was disproved by the author's experiment of one-sided destruction of the labyrinth, combined with de-afferentation, section of the posterior roots. Frogs used for this experiment showed the typical labyrinthian position of abduction of the posterior extremity, even after section of all posterior roots; therefore, the conclusion must be reached that the static and kinetic innervation is supplied by the axons of the anterior root cells.

Decerebrate rigidity does not increase metabolism. In decerebrate rigidity, the glycogen content of the muscles actually increases, although action currents can be shown to exist. The latter fact was demonstrated by the use of the string galvanometer. No actual work is done, however. The author does not believe that changes in the creatinin metabolism causes slowing of individual contractions. Changes in the carbohydrate metabolism do cause slowing of contractions as well as more permanent states of shortening. Muscles exhausted by repeated electrical stimulation in the absence of oxygen do not show increase of the creatinin content.

Swelling of muscle fibers is due to the formation of lactic acid. This causes increase in muscle tonus. Permanent increase in muscle tonus is not due to repeated stimuli and resultant repeated contractions. Such an increase in contraction is caused by the swelling of the muscle. No work, as before stated, is actually done. Various muscles can be dehydrated with greater or less difficulty as the case may be. The muscle of the oyster was used in a series of experiments. This muscle is made up of two distinct parts; a hard white smooth, and a glassy softer striated, muscle. These were separated. Both were then allowed to swell in dilute tenth normal hydrochloric acid, and dehydrated in 10 per cent. sodium chlorid. The smooth muscle could not be dehydrated to the weight it had before being treated with tenth normal hydrochloric acid, while the striated muscle sank below its initial weight.

The speed and contractile period of the red and white muscles of rabbits were compared. The difference in the speed of formation of lactic acid may

be looked on as the cause for difference in the contractile period of these muscles. The red muscles contract more slowly, and the contraction lasts longer. The white muscles contract more quickly but also relax more promptly. There is no difference in the swelling curves. The red muscle resists dehydrazation longer than the white, because lactic acid is formed more slowly and therefore is firmly bound to the protein molecule. The quicker lactic acid is formed, the quicker the muscle contracts. Quickly contracting muscles relax quickly because lactic acid is not firmly bound to their protein molecules.

The author next describes the various means that have been employed in measuring muscle tonus, and outlines the use of a mechanical apparatus of his own design which can be employed for measuring the muscle tonus of patients or laboratory animals. He discusses the higher mathematics of muscle tension curves, and cites algebraic formula used in obtaining such curves. From his own series of experiments, he draws the following conclusions:

The tension curve of a muscle with normal innervation is practically a straight line when the muscle is first stretched. Later this curve rises more abruptly as the opposing weights are increased beyond a certain point. There is a so-called "brake system," which functions even when the antagonistic muscles have been removed.

The action of this brake system persists under the influence of hypnotism and superficial narcosis. It disappears when proprioceptive stimuli are absent, as in the case of tabetic degeneration of the posterior roots. This brake system is therefore the result of a reflex which is conditioned by the physical resistance of the muscle toward being stretched. This reflex consists of two distinct reactions, a short quick contraction followed by continuous innervation, which seeks to keep the muscle in the shortened state reached by the first quick contraction.

The efferent path of this reflex is not dependent on the central neuron of the pyramidal tracts. The tension curve of a muscle shows no marked change in spastic conditions such as are encountered in cases of central hemiplegia and multiple sclerosis. On the other hand, the rigor of paralysis agitans is a direct result of this brake reflex. It is probable that the efferent path pursues an extrapyramidal course.

The proprioceptive reflex which causes the braking proceeds by way of the cerebellum. The path of the brake reflex must therefore run through other parts of the brain-stem, caudalward to the thalamus opticus. The axons of the anterior horns play the dualistic parts of supplying a way-station for both static and kinetic innervation.

After removal of the labyrinth in man an atonia of the homolateral quadriceps was observed. This was rapidly compensated, so that no difference between the muscles of the two sides was noted three weeks after the operation.

NEYMANN, Chicago.

AN HEPATICO LENTICULAR DEGENERATION WITH THE ACCOUNT OF A CASE AND THE PATHOLOGICAL FINDINGS. GEOFFREY HADFIELD, Brain **46**:147 (July) 1923.

Hall, on the basis of twenty-three cases collected from the world's literature, came to the conclusion, in spite of many secondary points of difference in the clinical picture, that progressive lenticular degeneration (Wilson's disease) pseudosclerosis (Westphal-Strümpell) and torsion spasm (Thomalla) may be grouped together under one term. He suggests the term "hepatico-lenticular

degeneration." The author describes a case which is not clearly a case of any one of the foregoing three conditions, but which presents characteristics of each.

The case is described in detail as follows: A school girl, aged 12½, had been perfectly normal until 8 years of age when, following a number of illnesses —measles, diphtheria and scarlet fever—she became delicate and needed considerable care. For about six months she was kept at home, and at the end of this time it was noticed that she was "very babyish." On her return to school she made slow progress, seemed queer to the teachers, and at 10 years of age it was noticed that she was awkward with her hands, while at 8 she had been able to knit well. She now became sensitive, but was mentally alert, affectionate and trustworthy. At about 11½ years, however, her mother noticed that the child's speech was thick, she was delicate, clung to her mother and had difficulty in holding things in her hands. She could not write, knit or feed herself. About this time also the mother noticed that the child's hands were shaking. Then in May, 1921, ten months before her death, the child developed a stoop, she kept her hands up to her face, and her involuntary movements became marked. Her mouth was now held open, her upper teeth were described as "sticking out," and the child salivated profusely. Her disposition changed to irritability and restlessness with violent outbursts of screaming. She developed a marked unsteadiness in her legs in November, 1921, and in December, her unsteadiness became so complete as to necessitate keeping her in bed, the symptoms described above becoming more marked, with remissions during which she seemed quite well. Her stoop was not extreme, her head shook in all directions, she twisted her face, and eyes, and when out of bed she would dance about the room all huddled up.

In February, 1922, she began to have convulsions, and was brought to the hospital.

Physical examination revealed the child in an attitude of semiflexion and showing no involuntary movements. When approached, her limbs became forcibly flexed and adducted, and she "shut up like a knife." Voluntary motion produced increased rigidity and clonic movements of the members in motion. She had a marked speech defect and was excitable. Memory was impaired. The liver was not palpable. The cranial nerves were essentially normal except for uncontrolled jerky movements when the lingual, sternomastoid or trapezius muscles were used. Sensation was normal. There was complete inability to perform a coordinated muscular act, extreme muscular rigidity, and walking was described as grotesque like a "dancing monkey." The reflexes were normal, the pupils dilated. After nine days in the hospital, she developed a series of about fifty convulsions, and died.

Necropsy showed a shrunken liver weighing 907 gm., with portal cirrhosis, and the surface studded with irregular granulations 2 to 12 mm. in diameter. On section, there was much fibrosis. Gross examination of the brain showed a loss of outline of the lenticular nuclei. Histologic examination revealed marked changes in certain areas. The putamen showed a definite selective atrophy of the large type of cell and a sparing of many of the small cells in which the atrophy, when present, was much more recent than in the large cells. The large cells were found swollen, and necrotic; the small cells showed an occasional neuronophagia. Chromatolytic changes in the small cells were less constant and had not progressed to the same degree as in the large cells. The putamen showed general and unmistakable gliosis. Prolonged search showed

two doubtful examples of the neuroglial giant cells reported by Alzheimer as specific of the neurogliosis in pseudosclerosis.

The caudate nucleus showed diffuse and advanced degeneration of the nerve cells, almost as constant as in the putamen. The changes were diffuse, and not focal. There was marked neurogliosis.

The globus pallidus showed generalized and diffuse chromatolytic changes in the bodies of the large motor cells, much less marked than in the putamen. Some gliosis was noted. The optic thalamus was essentially normal.

The cerebral cortex showed a prolific new formation of well-developed blood vessels in the frontal lobes, extending from the supraradiary layer to the junction of the gray and white matter. In the radiary layer, this new formation was developed to such a degree as to cause considerable distortion, interruption and degeneration of the nerve bundles. The vessels were of the caliber of capillaries and precapillaries. There was no dilatation or thrombosis of the vessels, and the perivascular spaces were large and roomy. Where these changes were most marked, one found neurogliosis and cortical chromatolysis.

The cerebellum showed no changes. The liver showed macroscopically and microscopically a multilobular cirrhosis. Cultures from the cecum, transverse colon, descending and ascending colon showed no pathogenic organisms.

In his discussion, the author calls attention to the fact that while clinically the case strongly resembles Wilson's disease, yet pathologically the case resembles pseudosclerosis more strongly. In comparing Wilson's disease and pseudosclerosis, the author points out that the muscular rigidity and the destructive changes in the lenticular nuclei are minimal in pseudosclerosis, while in Wilson's disease the rigidity is intense and the putamina often totally destroyed. "The histological changes in the case described consisted of a pathological neurogliosis which had entirely replaced the normal glia tissue of the putamina and caudate nuclei, and a selective necrosis of the large nerve cells of these nuclei. This change was sufficient to give rise to all the positive signs of Wilson's disease. It appears likely therefore that the histological changes described in the lenticular nuclei in the case reported, represent a stage in its functional destruction not far removed from the total degeneration found in well-established cases of Wilson's disease."

Pollock and Spielmeyer have reported findings similar to that of the author in the corpus striatum. Spielmeyer also found focal lesions in the cerebral cortex—intense nerve cell degeneration and marked neurogliosis. The author believes that the changes described in the cerebral cortex may be of the same nature as those found in the lenticular nucleus, but of more recent origin or of a less acute type.

ALPERS, Philadelphia.

THE HYPOPHYSEOBASAL AREA IN ITS RELATION TO THE PATHOGENESIS AND TREATMENT OF DIABETES INSIPIDUS AND POLYURIA. CHARLES E. DE M. SAJOUS, Am. J. M. Sc. **167**:679 (May) 1924.

In accord with recent literature emphasizing the need of a reconsideration of our whole attitude in relation to the functions of the pituitary body, the author turns his attention in particular to the part this gland takes in the production of polyuria.

Notwithstanding the views of a number of investigators that polyuria may be brought about by the tuber cinereum independent of the hypophysis, he believes the latter gland can be entirely excluded only with difficulty, and to

reconcile divergent opinions in this matter he assumes a neural structure and function for the posterior lobe "as the starting point of several sympathetic paths, one of which extends by way of the tuber cinereum, the bulb, cord and splanchnic nerves, and renal plexus to the vessels of the kidneys."

Results from animal experimentation show the existence of a nuclear aggregate at the base of the brain which may carry on all vegetative or somatic functions independent of the cerebral hemispheres proper, included in which is the posterior or neural lobe of the pituitary, whose function he likens to one of the cerebral motor areas. He terms it the hypophyseobasal area, and offers evidence that, resembling the cortical gray substance, it contains true nerve cells and fibers; the latter traversing the infundibulum and tuber cinereum to the so-called nuclei tuberi in the tuber cinereum. The infundibulum in the mature subject is not simply a hollow outgrowth from the third ventricle as sometimes supposed, but rather a solid stalk affording sufficient pathway for these fibers.

The possible influences of the hypophysis on the body at large include not only the results from stimulation of the organ itself, but also those produced by even slight interference with the impulses through the hypophyseorenal path, as described by the author, and diabetes insipidus or other disorders may result from lesions (demonstrable at necropsy) anywhere throughout its course. Hence, twelve cases are described with lesions of the posterior lobe per se; six with lesions in both lobes and in the infundibulum; two in which the infundibulum and tuber cinereum were involved; seven, the bulb; and four with lesion of the spinal cord and splanchnic nerves. These and other cases are offered as evidence that the posterior lobe of the hypophysis and other structures of the hypophyseorenal tract exert a constant physiologic influence on the renal functions. As for the methods by which the flow of urine is increased, the writer states that the renal arterioles are dilated by the vasodilator terminals of the sympathetic through the splanchnic nerves and semilunar ganglia, causing congestion of the kidneys.

He concludes that any condition intererfering with the transmission of impulses through the hypophyseorenal path to the renal vascular system may produce polyuria. Pregnancy as a cause is explained by pressure of a congested anterior lobe on the nervous elements of the posterior lobe. Pressure is also the cause in acromegaly, hypophyseal tumors and structural disorders in the immediate vicinity. Dilatation of the third ventricle comes under this group, as does also epidemic encephalitis and meningitis by involvement of the hypophysis and its overlying structures.

To explain why falls on the head, concussions, basal fracture and violent emotions may cause diabetes insipidus, the writer further emphasizes the similarity of the neural structure of the hypophysis to the cerebral cortex as observed by histologists and by his own studies, and gives reasons which lead him to refer to this organ with its nervous connections as a somatic brain or a sensorium commune. This is capable of reacting to fright, shock, anger and other emotions. Cutaneous sensibility, whose proper center is still in doubt, may be located here. Special types of pain sense, touch, hearing and taste are known to be connected with the hypophysis. The source of the production of the many peripheral symptoms observed in hypophyseal disorders is similar to the cause of polyuria, namely, passive vasodilatation in the tissues which are the seat of the symptoms. There is also evidence to show that the hypophysis is capable of receiving sensory and giving out motor

impulses—the inward impulses traveling through the median fillet and the motor impulses to the vessels by the spinal sympathetic system.

The extract of the posterior lobe as usually given for the relief of polyuria does not contain the secretion of the organ, but only pharmacologic products such as organic phosphorus and epinephrin which reduce the caliber of the arterioles, restore the normal renal circulatory equilibrium and thus diminish the flow of urine. However, as its action is ephemeral, the author, in one of his cases (a child, aged 12, with pituitary tumor) gave desiccated hypophysis 1/10 grain (0.006 gm.) and purified extract of ergot (ergotin, Merck) 1 grain (0.06 gm.) in capsule, orally, three times daily (the ergotin being used to reinforce the other). His results were successful over a period of two years. Enuresis in children due in some instances to adynamia of the hypophyseobasal area may be benefited by such treatment.

Removal of both the anterior and posterior lobes of the pituitary in dogs was usually followed by death, the animals becoming somnolent and dying, in from twenty-four to thirty-six hours, from respiratory failure. The path from the hypophyseobasal area to the suprarenals, the latter sustaining tissue oxidation, as traced by the author some years ago, would explain the cause of death in these animals, while in those which survived this operation the remaining uninjured part of this nerve tract was sufficient to carry on the vital processes concerned.

BARRETT, Philadelphia.

A CASE OF ACUTE VERBAL APHASIA FOLLOWED THROUGH THE VARIOUS STAGES OF RECOVERY. HENRY HEAD, Schweiz. Arch. f. Neurol. u. Psychiat., Festschrift f. Constantin von Monakow **13**:313-324, 1923.

Most authors seek to bring the initial manifestations of a brain lesion into direct connection with the anatomic alterations found in the neighborhood of the injury which consist principally of certain circulatory disturbances, edema, and secondary inflammatory changes. Monakow on the other hand speaks of a condition of diaschisis. The local destruction of brain tissue causes a cessation of function of neighboring and closely allied neuronal systems. This extends to the termination or to the origin of each system of fibers involved in the local lesion. Those paths of innervation suffer most severely which subserve the more complex activities or those which are less organized by practice. Restoration of function depends first on the recovery from pathologic processes of a grossly organic nature, secondly the passing away of diaschisis, and lastly the gradual assumption of compensatory powers of uninjured portions of the nervous system.

The more a movement is directed toward a definite object and the later it is acquired in the life of the individual, the greater the probability that it will be represented in the cortex although the actual forces putting it into action are of subcortical origin. Movements of expression comprise the largest number of diverse elements. Among these factors is the symbol, a gesture standing for the widest possible combinations of stimuli. It is obvious that no topographically distinct center exists for speech or for any such complex acts as reading and writing.

Clinically speaking, the course of a case of aphasia due to an acute nonprogressive lesion can be divided into three periods: First we observe a period of shock, reinforced by secondary structural changes such as edema. This may pass away within a few weeks, when the phenomena of diaschisis become

revealed. They do not express solely the lost physiological activity of the destroyed tissues but represent a wide-spread disorganization of cerebral function. After many weeks or months these may pass away to reveal residual symptoms which represent a permanent disintegration dependent on tissue destruction.

The following case was that of a woman 56 years of age, who for six years had suffered from Jacksonian seizures beginning in the right hand. On May 20, 1922, after a seizure, she found that she could not speak for several hours. Ten days later the only abnormal physical sign was loss of reflexes over the right half of the abdominal wall. On June 20, an endothelioma arising from the dura mater, which had severely indented the substance of the brain where the inferior frontal meets the precentral fissure, was removed so that not the smallest fragment of cortical tissue adhered to the tumor. The operation was followed by complete verbal aphasia and right hemiplegia. She improved rapidly day by day. At first her speech consisted of "yes" and "no," but she failed to comprehend the full significance of questions. Within the first six days "yes" and "no" were employed appropriately. She used a few ejaculatory words, spoke extremely slowly, and slurred the syllables. By the seventh day marked improvement had taken place and she could name ten of eighteen objects. Writing and spelling were defective. Printed commands necessitate recognition of verbal differences and were carried out with more difficulty than were verbal commands. In writing it was noted she was dictating to herself. Three weeks later she still had difficulty in writing; the spelling was poor and the letters were badly formed. Four months after the operation she could write names well and this test was the last to be carried out correctly. She arranged blocks imperfectly at first but thirty days after the operation blocks were arranged in due sequence. She then wrote perfectly on dictation. Copying suffered equally with spontaneous writing because she uttered each word to herself before she wrote it. The clock test seemed to be guided by whispered audible or inaudible words. She had great difficulty when the command was printed in railroad time. She had not the slightest difficulty in imitating movements reflected in a mirror but reversed the response in seven out of sixteen trials. The power to carry out oral commands was the first to recover. Here the words were enunciated by the observer and it is only necessary that the patient remember them long enough to execute the required movements. Next in order of difficulty came the translation of printed orders into action. During the course of recovery the power of writing passed through analogous stages. Four months after the operation, the only defects were hesitation and slight uncertainty in forming certain letters.

In this example of verbal aphasia the acts of speaking, reading, and writing were all affected at first. Apart from the difficulty of finding correct verbal forms there was no loss of ability to name familiar objects. As she regained her power of using language those accomplishments returned first which were least dependent on accurate word formation. Of all the tests those which necessitated writing were the most severely affected. Long after ordinary tests failed to show that her use of language was defective the patient was conscious of something being wrong and the observer could detect faults in articulation. The physical signs followed the same rules of recovery as the aphasia. The details of this case are to be published in full in the author's book on "Aphasia and Kindred Disorders of Speech."

WOLTMAN, Rochester, Minn.

PERSONALITY CHANGES IN CHRONIC ENCEPHALITIS. DR. MAX MEYER, Klin. Wchnschr. 3:137-141 (Jan. 22) 1924.

Bonhoeffer has attempted to classify the psychic manifestations in the acute stages of encephalitis as exogenous reaction types. In the chronic states of encephalitis in which the motor field has been but slightly affected, the psychic manifestations, when present, are more often of a neurasthenic type with variable fatigability, difficulty of attention, somnolence and a rather characteristic moroseness of speech without definitely associated intellectual disturbances. This psychic apathy and depression may continue long after all physical signs of encephalitis have disappeared and may even show a tendency to periodic recurrence. The depressive moods are more frequent than the euphoric-hyperthymic reactions.

In the more severe forms of psychic changes in the akinetic types of chronic encephalitis, there develops an increasing slowness of speech, and all mental processes appear to run parallel to the motor phenomena. At times apathy may become profound, and Hartman has termed it "motor dementia." Such persons accept the occurrences of their environment without any attempt to analyze or investigate them. It has been stated that this indifference or loss of affect is a primary difficulty. The author is of the opinion that the affective disturbances result from the generalized akinesia. The depressive reactions are by far the more common, and these may be so marked as to lead to suicide. One physician well expressed himself when he said that his patients resembled a "burned-out crater." Euphoria is much rarer, although in cases without motor disturbances, witzelsucht may be present to some degree.

In one case in which the parkinsonian syndrome was marked, the patient showed distinct fluctuations in behavior. There would be periods of apathy and depression with irritability and carelessness. The patient would go to bed with his clothes on and refuse to see visitors; then there would be periods of activity in which he showed extreme interest in the surroundings and marked social activity. Finally, during a period of depression, suicide was committed.

Affect epilepsy and outbursts of anger are not uncommon, especially in the hyperkinetic disturbances. Psychomotor unrest and various other neurasthenic complaints may be present. These patients have a tendency to lose interest in their work or surroundings. Mannerisms and stereotyped movements are rarely noted, but occasionally somnambulistic activities present themselves.

A somewhat similar group of cases are the behavior disturbances in children following a latent period after an encephalitis. In the milder forms of this disturbance, psychomotor unrest, such as scratching or picking of the nose or skin, spitting, even asocial acts with impulsive behavior and pyromania may occur. In some of these children, besides behavior disturbance, changes in sleep rhythm and respiratory phenomena occur. Thus a girl, aged 14, who in January, 1920, had encephalitis, in the fall of 1921 developed attacks of anxiety with outbursts of anger in which she would curse, pound the bed, salivate and spit on the walls. With this there would be a respiratory apnea. This respiratory difficulty might occur without psychic disturbance. After several months of variable therapy, the attacks became less severe. Such latent periods are not uncommon. It is especially true that this type of affection in children is usually unassociated with the parkinsonian syndrome. In spite of the personality changes, there appears to be no distinct intellectual weakness. The so-called "'tween brain" disorders are commonly associated

with behavior disturbances. Under this heading internal secretory disturbances, such as marked increase of weight, polydypsia, polyuria and sleep disturbances are noted. Probably functional disturbances are the result of some affection on the floor of the third ventricle. Such syndromes may also occur in adults. MacNeil Donald reports the case of a man, aged 25, who, following an encephalitis, showed a marked character disturbance. He had been upright and in every way reputable. He began to fabricate, became untruthful and morally delinquent, but showed no definite impairment of intelligence. He presented a characteristic picture of dystrophia adiposa genitalis.

This change in moral character, while uncommon in adults, has also been observed by the author in one case. A bank clerk who had always been very industrious, following encephalitis developed a marked parkinsonian attitude. With his physical change, he became disinterested in his work, was suspicious of his wife, stated that she was putting poison into his food. He incurred debts without any indication of anxiety and finally was arrested for exhibitionism. While in the hospital he improved, but as soon as he was returned to his home his symptoms returned.

The author in reviewing the cases, and in discussing the psychic manifestation following encephalitis, suggests that these mental changes are probably organic in nature, and that they are a counterpart of the motor disturbances following encephalitis. It is possible that as our conception grows we will become more and more inclined to look on such character changes as organic and will refuse to be satisfied with a psychogenic explanation.

MOERSCH, Rochester, Minn.

GENERALIZED CISTERNAL ARACHNOIDITIS SIMULATING CEREBELLAR TUMOR: ITS SURGICAL TREATMENT AND END-RESULTS. GILBERT HORRAX, Arch. Surg. **9**:95 (July) 1924.

In this communication, Horrax calls attention to the condition of cystic arachnoiditis occurring about the basilar cistern at the base of the brain. This condition produces the same effect as does a tumor in this region, and the resulting hydrocephalus with increased intracranial tension causing papilledema may demand prompt surgical relief if vision is to be preserved and discomforts alleviated. Cystic arachnoiditis may be encountered over almost every portion of the cerebrum, cerebellum and spinal cord. These localized lesions have been classified by some authors as pseudotumors.

Quincke, in 1893, first described the condition as a meningitis serosa, dividing the cases into acute and chronic types. Increased intracranial pressure plus fever were the most prominent symptoms. The differential diagnosis from tumor, abscess and purulent and tuberculous meningitis had to be made in each instance, and sometimes was extremely difficult to make. Such differentiation was determined largely by examination of the cerebrospinal fluid as obtained by lumbar puncture, since clinical studies were always confusing. If the fluid contained no pus or tubercle bacilli, but showed an increased amount of albumin, and if all symptoms were relieved for a considerable period of time after one or more punctures, the diagnosis of serous meningitis was believed to be established. A few cases came to postmortem, but these showed no changes in the meninges, a fact that the author did not regard as invalidating the diagnosis.

The generalized condition, however, is apparently quite a distinct entity. In the cases that form the basis of this study, such examination as could be made at the time of operation revealed a thickening and grayish opacity of

the arachnoid forming the large posterior cistern over the cerebellum, the cistern often being greatly enlarged. In the five fatal cases, the arachnoid thickening of the basilar cisternae as well as that overlying the cerebellum was present also. The process produces a blocking of the cerebrospinal fluid system, giving an internal hydrocephalus, and the filling up of the cisternae of the posterior fossa dominates the picture. We have therefore termed the condition generalized cisternal arachnoiditis. The neurologic findings almost invariably give the localizing features of a cerebellar lesion simulating tumor.

The results in a series of thirty-three cases are presented which, from the standpoint of clinical diagnosis, closely resembled tumors of the cerebellum. All the patients reported were operated on with the presumptive diagnosis of tumor, and in none was a tumor found. Five of these patients died, two of them within a few days after operation, and the other three anywhere from several months to two years later. Of the four on whom necropsy was obtained, all showed histologically inflammatory thickening of the arachnoid membrane forming the cerebellar and basal cisternae. Of the twenty-eight patients who survived, all have been improved or were well from one to nine years after operation. In the living patients, there was disclosed at operation over the cerebellum a greatly dilated posterior cistern with its thickened arachnoid membrane, which contained pent-up cerebrospinal fluid under pressure. In a few instances, a piece of the arachnoid wall was removed for histologic examination, and these specimens showed nothing more than the slight thickening that was evident grossly. Such evidence, although not so conclusive as might be desired, coupled with the subsequent improvement or entire relief of symptoms over long periods of time, has convinced us that the condition was not tumor but arachnoiditis, as surmized at the time the posterior fossa was exposed.

Apparently in the fatal cases which were carefully studied histologically an encephalitis existed, together with the basilar arachnoiditis. Fever and leukocytosis were rarely seen, which differentiates this condition from the acute meningo-encephalitis. No serologic or bacteriologic evidence of syphilis or tuberculosis were noted.

At times a basilar arachnoid cyst of the lateral recess may overlie a tumor. This may be the underlying cause in some of the patients that were operated on only one or two years ago, although careful examination failed to reveal the neoplasm. Except in the five instances in which the arachnoiditis seemed definitely to follow otitis media, the etiology of the condition is obscure. The treatment indicated is suboccipital craniectomy, and in the absence of a tumor, the infiltrated arachnoid membrane should be opened widely and its contents evacuated. In the large majority of patients, this was sufficient to affect an entire relief of the pressure symptoms, at least for the varying lengths of time they have been followed. Lumbar puncture was not employed as a diagnostic measure, for it was believed that the risk of a fatality from foraminal herniation of the cerebellum was too great in patients with increased intracranial pressure.

GRANT, Philadelphia.

THE SYNDROME OF INFLUENCE. A. CEILLIER, L'Encephale **19**:152-162 (March) 1924; 225-234 (April) 1924; 294-301 (May) 1924; 270-301 (June) 1924.

In this paper, the author presents a detailed study of ideas of influence. Patients presenting the syndrome of influence refer their thoughts and acts to some foreign personality which is often stated to be a spirit. Ceillier denies

the existence of an "essential psychosis of influence" but maintains that the phenomena are symptomatic. This syndrome may occur as a manifestation in any psychosis, although at times there is no apparent underlying psychopathic state. The latter, or primary form of this syndrome, usually occurs in the third decade. It is more common in women, possibly, as is suggested by the author, because they not only are more apt to have sexual conflicts and physiologic disorders, but are also more suggestible than men. It is rare in childhood, and when it occurs in old people, it is ephemeral.

The phenomena are the result of two factors in combination usually. First, there is the patient's claim that his acts and thoughts are due to foreign influence—hypnotism, occult knowledge, etc. Second, there is an automatism associated with dissociation of the conscious personality.

The phenomena of automatism are numerous, and are not recognized by the patient as belonging to his own individuality. Visions, abnormal alterations of emotion or mood, compulsions, "internal conversation," automatic acts —such as automatic writing and speech, and pseudohallucinations are illustrative. The author defines pseudohallucinations, which are frequent in the syndrome of influence, as being characterized by: (1) their appearance without the will of the patient, (2) the inability of the patient to rid himself of them, (3) the usual passive attitude on the part of the patient toward them, (4) lack of exteriorization—that is, the patient does not localize them in space but as "in his mind." Mental conversations with voices are common and are good evidence of dissociation. True sensory hallucinations are rare. However, somatic sensations, especially of the genitals, are common.

The ideas in the syndrome of influence may be contrary to the conscious moods and thoughts of the patient, and give clues to subconscious trends. True automatic acts, which are those done *in spite of* a patient's will, are rare. When they occur they strengthen the patient's impression of being under an influence. The emotional state depends somewhat on the nature of the ideas present, as well as on the degree of resistance exercised by the patient. The syndrome may be classed clinically according to the type of delusions: (1) protection, (2) persecution, (3) erotomania, (4) spiritualistic, and (5) mystic and demoniacal. In the spiritualistic form the symptomatology is rich, and automatic phenomena are well developed. This form may occur in persons without any previous interest in spiritualism. However, spiritualism seems to favor the development of psychoses of influence. In addition to frauds and mediums who are really sick, there are some predisposed persons who after training, develop automatic writing. Finally, some seem to have a special constitutional predisposition—a weakness of will, great suggestibility with a need for relief or comfort, and a certain excess of reproductive imagination.

The syndrome may also be classified by the degree of automatism and the character of the fundamental pathologic phenomena: (1) a form of interpretation in which the patient recognizes his acts and ideas as abnormal, but explains them as due to some foreign influence acting on his mind; (2) with disintegration of the conscious personality, and (3) delusions of possession.

The relations of the state called psychasthenia to the syndrome of influence are especially interesting, because they illustrate the transition from a neuropathic to a delusional state. Moreover, certain of these cases are amenable to psychotherapy and can be cured. The abulia, feeling of inadequacy, apathy and feeling of unreality so common in psychasthenics are predisposing factors.

Obsession states are closely related to syndromes of influence and both are the results of a psychologic automatism. The "obsessed" patient, however, retains

some insight and will understand the mechanism of his illness. But although he resists the idea of obsession as absurd, he judges it to be imposed by an external force or influence.

The prognosis depends not only on the presence of an accompanying psychopathic state, but also on the nature of any discoverable conflicts. Close psychologic study of the patient is always necessary. Physical factors and causes, especially endocrinopathies, must be searched for. In medicolegal work, one must be on the lookout for this syndrome—as well as its simulation, when the person under charges will pretend that his acts have been committed under an influence.

HYSLOP, New York.

CORTICOTHALAMIC CONNECTIONS OF SOME AREAS OF THE BRAIN IN THE RABBIT. J. M. DE VILLAVERDE, Libro en honor de S. Ramón y Cajal **1**:591-624, 1922.

Different portions of the cortex of the posterior region of the brain were destroyed by means of the thermocautery. The animals were killed fifteen days after the operation. Marchi slides were prepared. Sections were cut frontally.

In the first rabbit, the thermocautery penetrated through Brodmann's area 18, near area 29. The lesion destroyed part of area 18 and deep regions of areas 29*d* and 29*c*. A similar lesion was produced in the second animal, with destruction of area 29*d* and total destruction of area 29*c*, producing a small amount of disorganization in area 18. In the third rabbit, the lesion was extensive. The chief lesion was situated between Brodmann's areas 22 and 21, which were almost totally destroyed; area 17 was probably injured. The lesion was very deep, destroying both the gray and white matter, and reaching the ventricle. Although a number of cortical areas, as for instance areas 29 and 18, were intact, the place through which their projection systems pass in order to reach the thalamus was destroyed.

The secondary degenerations produced by these lesions are described in detail, and the findings of Winckler, Berl and Probst are reviewed and discussed. The results of Villaverde's studies show that abundant fibers from the hemisphere reach the thalamus through the most caudal portion of the internal capsule, passing around the caudate nucleus. It is believed that they cross through the reticular nucleus, in which a few fibers and collaterals end, and that they are distributed to the other thalamic nuclei. The further course of the fibers ending in the reticular nucleus could not be established. In less degree, fibers from the hemisphere also reach the thalamic nuclei through the optic radiation of the thalamus. Degenerated fibers in the radiation are most abundant in those cases in which the lesion of the cortex was very extensive. A part of them reaches the superior colliculus, while others end in the superficial nuclei of the thalamus (dorsal part of the lateral external geniculate body and lateral nucleus).

Degenerated fibers were constantly found in the external geniculate bodies of the three rabbits, but their numbers were variable, according to the extension of the lesion. They were more numerous in the third rabbit in which the temporal region was injured. In this brain, the secondary degeneration of the dorsolateral geniculate is chiefly located in segment *a* of the ganglion (external segment). In deep regions (segment *b*), degenerated fibers are less frequent. This condition contrasts with that found in the second rabbit in which the lesion, less extensive, was restricted to area 29 and a few of the more dorsal regions of area 18. Secondary degenerations were scarce in segment *a* and

were absent in segment *b*. In the first rabbit, the dorsolateral geniculate ganglion contains abundant degenerated fibers which enter the ganglion along with the bundles of fibers from the optic radiation. In deep regions of the ganglion, there are also numerous degenerated fibers. In this animal, a part of area 18 was destroyed. From these facts it is concluded that in the dorsolateral geniculate ganglion there is not separation of the corticothalamic fibers in segment *a* and segment *b*, as described by Winckler. Villaverde thinks that this ganglion represents a single body in which area 18 is chiefly represented.

The ventrolateral geniculate ganglion in the first and third rabbits shows abundant degenerated fibers, which travel toward its dorsal region. Similar conditions are found in the dorsolateral geniculate ganglion, whereas in the second rabbit degenerated fibers are scarce in both geniculate ganglia. It is suggested, therefore, that the ventrolateral geniculate is not a terminal center for the fibers, but that the latter cross through it.

The lateral nucleus of the thalamus in the three rabbits shows secondary degenerations. The fibers reach its deep regions through the lamellae of white substance of the thalamus, which are a posterior prolongation of the white matter of the reticular nucleus. Villaverde tentatively advances the hypothesis that the lateral nucleus receives the fibers from the retrosplenial area of Brodmann, recently studied by Cajal, who finds in it a peculiar cyto-architectonic and myelo-architectonic. According to this author, area *29d* in the rabbit does not have the boundaries assigned by Brodmann, but extends outward in the dorsal surface of the hemisphere. This fact has been confirmed by Villaverde in the second rabbit, in which area *29d* was destroyed, causing, it was thought, a small amount of disorganization in the most internal region of area 18. The absence of a lesion in the latter is evidenced by the small amount of degenerated fibers in the dorsolateral geniculate ganglion.

NONIDEZ, New York.

THE INFLUENCE OF THE CEREBRUM AND CEREBELLUM ON EXTENSOR RIGIDITY. W. P. WARNER and J. OLMSTED, Brain **46**:189 (July) 1923.

Sherrington pointed out that decerebrate rigidity in the experimental animal resulted from decerebration at the level of the mid-brain. He further showed that on the removal of only one hemisphere, the rigidity was inconstant and nonpersistent, but was more marked on the ipsolateral side. He showed further that decerebrate rigidity is abolished if the continuity of the proprioceptive reflex arc is abolished. He considered the cerebellum as an unessential part in the production of decerebrate rigidity, since this condition often remained after the cerebellum was removed.

Thiele, however, on the basis of experimental work, concluded that the production of decerebrate rigidity is due, not to a removal of the cortical influence, but to a thalamic influence. He found also that extensor rigidity followed ablation of certain parts of the cerebellum, and concluded that the cerebellum exerted an influence on decerebrate rigidity. Rigidity could be destroyed by destroying the dentate nucleus, or by cutting the trapezal fibers in the pons. Weed concluded that the center and source of decerebrate rigidity lay in the red nucleus. Magnus and Bazett and Penfield, however, deny the statement of Weed, since rigidity persists when the brain stem is transected posterior to the red nucleus. Bazett and Penfield locate the center for decerebrate rigidity at the level of Deiters' nucleus.

The object of the authors' investigation was to discover if possible the exact location of the inhibitory pathway, and the effects produced by its destruction

at various levels. All the experiments may be stated briefly as follows: Ablation of the motor cortex of the cerebral hemispheres did not result in the production of decerebrate rigidity. Rigidity occurred only when the frontal areas were destroyed. The destruction of only one frontal area, or the interruption of fibers arising from the frontal lobe, and running in the internal capsule anywhere in their course down to the level of the anterior corpora quadrigenima, resulted in the production of contralateral rigidity with slight ipsolateral rigidity. After the destruction of the frontal area, successive sections through the entire hemisphere did not increase the rigidity. The rigidity disappeared when the section passed through the posterior corpora quadrigemina. The extensor rigidity could be inhibited by stimulating (1) the cut surface of the frontal lobe, (2) the mesial side of the anterior limb of the internal capsule, (3) the mesial side of the cerebral peduncle, (4) the brain stem just posterior to the anterior corpora quadrigemina. In the first three cases inhibition occurred on the contralateral side, and in the last case, on the ipsolateral side.

"The experimental evidence therefore shows that there are tracts in the brain of the cat carrying impulses which tend to inhibit tonic extensor activity, and that decerebrate rigidity results when the influence of the tracts is cut off. The tracts have their origin in the frontal lobes of the cerebral hemispheres. . . . From the frontal lobe the tract passes through the mesial part of the internal capsule and extends caudad past the level of the anterior corpora quadrigemina. It then, in the greater part at least, crosses to the other side of the brain stem to enter the cerebellum by way of the middle cerebellar peduncle. The tract thus marked out experimentally agrees in general with the so-called 'cortico-ponto-cerebellar' path."—(Ranson)

ALPERS, Philadelphia.

TOXIC AMINS PRESENT IN DEMENTIA PRAECOX, ABSENT IN MANIA AND MELANCHOLIA. V. M. BUSCAINO, Rassegna di Studi Pshiatrici **12**:4-5, 1923.

This paper represents a supplement to the first report made by Buscaino at the meeting of the Italian Neurological Society in Florence, October, 1921, in which he described some abnormal amins in the urine of patients with dementia praecox. Since then he has made several other reports on the same subject, in which his silver nitrate reaction was described and his findings were confirmed. In this paper, he states that by using an improved method, he has succeeded in obtaining 100 per cent. positive reactions from 55 per cent. obtained at the time of his first report. According to the author, the appearance of a "dark black" reaction in the urine of patients with dementia praecox indicates the presence of special toxic substances capable of injuring in a particular manner the central nervous system of such patients. These abnormal substances (amins) are found in cases of amentia, but are never found in cases of mania and melancholia without febrile complications. These facts are substantiated by more than one thousand reactions obtained in twenty cases of dementia praecox and five cases of mania and melancholia. The general conclusions of this paper are:

(1) The improved silver nitrate reaction with the use of heat shows in the urine of patients with dementia praecox, in all the cases examined, the presence of decidedly abnormal amins (black type precipitates).

(2) The elimination of such abnormal products is more rapid at the onset of the illness. In general, it appears in an intermittent way without regularity,

in old as well as in recent forms, in hebephrenic, catatonic and paranoid forms, in depressive syndromes with schizoid signs and also in episodes of schizophrenic exaltation appearing in the course of a typic mania. Such elimination does not depend on febrile manifestations or on tuberculous infections, but depends on the absorption of certain toxic products (amins with imido-azolic nucleus) which are formed, most probably in the small intestine, through specific, chronic processes (infections?). The absorption is facilitated by conditions of hepatic insufficiency.

(3) The most frequent types of precipitate are in general, the dark brown and the "dark black." The last one, which is due to the presence of compounds of beta-imidazolylethylamin, or histamin, is more constant during the first period of the illness. In the catatonic forms, the "black greenish" and the "black earth" precipitates are more frequent; the same fact is observed in extrapyramidal syndromes (personal cases of "post-encephalitic" amyostatic forms, Wilson's syndrome, parkinsonian and chronic cerebellar syndromes).

(4) Dementia praecox is therefore an acquired disease (occurring in a brain often biologically weak), toxic in relation to the brain and due to the presence in the circulatory system of particular substances (amins), especially in compounds of histamin. By injecting histamin chlorid into rabbits, typical substances of disintegration appear, after a long time. This is a form of alteration frequently encountered in the encephalon of dementia praecox cases.

(5) In patients with true manias and in those suffering from melancholy without actual febrile manifestations, the amins here described are not found in the circulation.

(6) The hot silver nitrate reaction is therefore of diagnostic value in the differentiation of schizophrenic and true forms of mania and melancholia.

NACCARATI, New York.

ANATOMICAL STUDY OF TUBERCULOSIS OF THE NERVOUS CENTERS. I. BERTRAND and G. MEDAKOVITCH, Ann. de méd. **15**:419, 1924.

The authors note that the diagnosis of cerebral tubercles is difficult, as these tubercles do not resemble those found in other organs. They may resemble meningeal sarcoma, syphilitic lesions, or abscess due to the usual pyogenic organisms. Several years ago the authors presented a case of cerebral tubercle before the French Société de Neurologie; it was a single fibrous tubercle that resembled a fibrosarcoma, and the diagnosis of tuberculosis was questioned by some of the members of the society. Since that time they have made a special study of tuberculous lesions of the central nervous system. Their material covers thirty-one cases. They have developed special staining methods for demonstrating the tubercle bacilli in anatomic sections, and for the study of the histologic changes in the nervous centers due to the tubercle bacilli. As a result of these studies they have found that in meningeal tuberculosis the characteristic lesions are diffuse, either serocongestive, necrosing or caseous. The latter form is found only in adults. Meningeal lesions do not show tubercles.

Cerebral tubercles may be divided into two chief groups—corticomeningeal and central, the former being the more frequent. These are further subdivided. The corticomeningeal forms include: (1) Pseudogummatous plaques. As a rule, there are no giant cells or typical tubercular centers in these lesions, and differentiation from gumma must be made by demonstrating the presence of tubercle bacilli. (2) Multiple cortical tubercles affecting only the gray

matter, from 3 to 5 mm. in diameter, not extending over the whole cortex but usually confined to one lobe of the cerebrum. (3) Abortive subcortical tubercles with sclerocaseous centers containing only a few tubercle bacilli, with no evidence of lymphoid metaplasia or perivascular inflammation. (4) Fibrous, pseudosarcomatous tubercle, usually a large isolated tubercle, which because of its adherence to the meninges may suggest a meningeal tumor, such as fibrosarcoma; histologically, however, this lesion is a true tubercle with caseous centers and giant cells. The central forms include: (1) diffuse encephalitis, resembling in many respects glioma with ameboid cells; (2) purulent abscess with a structure almost similar to that of the ordinary type of brain abscess, the center filled with degenerating leukocytes, the mononuclears containing the tubercle bacilli in large numbers; (3) the central fibrocaseous form, homologous to the pseudosarcomatous fibrous tubercle of the cortical type and showing the structure of the tubercle.

In tuberculosis of the cerebellum, the origin of the lesion is practically always at the meninges, and an extension of the meningeal involvement. Only four cases of this type were observed; in two, the lesions were fibrous and might have been operable; in one case, one of the cerebellar hemispheres was almost completely destroyed; in the fourth case, there was an inflammatory lesion developing in the course of extensive visceral tuberculosis.

Tuberculosis of the pons varolii is located chiefly at the base of the pons and especially in the pyramidal tract; the lesion is typically a tubercle surrounded by an area of degeneration, and almost invariably another tubercle is to be found in one of the hemispheres of the cerebellum.

Intramedullary tubercle is rarely found—in only two cases in the authors' series; the giant cells present were of vascular origin.

DAVIS, New York.

ETIOPATHOGENESIS OF THE HEPATOLENTICULAR DEGENERATIONS: ALCOHOLISM AS THE CAUSE OF HEPATONEUROGLIA DEGENERATION. L. INSABATO, Arch. Gen. di Neurol-Pschiat. e Psicoan. **4-5**:1-2 (March) 1924.

Insabato starts with the analysis of a case of alcoholic pseudoparalysis reported by him, two cases reported by Demole and Redalié and a case by Frigerio, calling for a revision of the whole conception of hepatolenticular degeneration. The histopathologic findings of his case were: Laennec's atrophic hepatic cirrhosis associated with profound alteration of the neuroglia in the whole encephalon, the cerebellum included, consisting in proliferation of protoplasmatic glia with formation of giant tortuous nuclei and polymorphonuclear elements similar to the giant glial cells found by Alzheimer in pseudosclerosis. The two cases of Demole and Redalié, reports of which were published six months after the author's report, were those of alcoholic patients who presented extrapyramidal symptoms, dementia, hepatic cirrhosis and glia alterations, of the Alzheimer type. The case of Frigerio was also that of an alcoholic who presented extrapyramidal and hepatic symptoms. From these cases and from the analysis of the literature on the subject of hepatolenticular degenerations, the author comes to the conclusion that such syndromes are related to alcoholism. He points out that in Wilson's disease, pseudosclerosis and torsion spasm, there exist states of transition in the symptomatology and in the anatomic findings, but he denies that the cirrhosis with large knots is a characteristic anatomopathologic entity of the hepatolenticular degenerations. These large knots are macroscopic in constant results of histopathologic

reactive, nondegenerative processes. The only constant characteristics found in all are formations of new connective tissue and parvocellular infiltration. In general the differential features in Laennec's cirrhosis and cirrhosis with lenticular degeneration are vague and uncertain. The absence of ascites does not characterize the cirrhosis of hepatolenticular type. There are many alcoholic cirrhoses which are found only at necropsy. In thirty-seven necropsies on chronic alcoholic cases, made by the author, ascites was found in only five cases. The atypical glial reaction of Alzheimer-Hosslin is not pathognomonic of the pseudoscleroses, nor of the other hepatolenticular degenerations. It may appear in all the morbid processes in which the liver is the seat of interstitial inflammation.

The characteristic neuroglia changes are not always nor especially in the basal ganglia, and the author points out that the name hepatolenticular degeneration must be changed to that of hepatoneuroglia degeneration. The hepatolenticular degenerations should constitute a subdivision of the whole group to indicate the cases of Wilson's disease and torsion spasm.

The author discusses the important question of heredity, and recalls in this connection the researches of Buscaino, who showed that in dementia praecox, postencephalitic syndromes, amentia, Parkinson's disease, and other conditions, particular toxic substances (aminic bases) are found, which possess a special tendency to produce, by injuring first or contemporaneously the liver, reactive phenomena in the basal ganglia, leading to the formation of large oligoplasmatic glia nuclei. His general conclusions are in favor of the hepatic lesion preceding the encephalic degeneration, and also the toxic pathogenesis of the greater part of the hepatolenticular degenerations.

NACCARATI, New York.

RESTITUTION AFTER A LOCALIZED BRAIN DEFECT. KURT GOLDSTEIN, Schweiz. Arch. f. Neurol. u. Psychiat., Festschr. f. Constantin von Monakow **13**: 283-296, 1923.

It has been known for a long time how little a complete hemianopsia may disturb an individual. The field of vision in these instances, therefore, must have the same general function as that of the normal eye. A normal field of vision extends in all directions around the point of fixation; the size of this field varies according to the territory we find it necessary to cover at the given moment. The field of vision of a hemianoptic person behaves almost similarly. He has not a half field of vision but a field that extends in all directions around a central point, this point appearing to him to be directly ahead. Fuchs has shown that the point of clearest vision lies within this field. This subjective point of clearest vision cannot correspond to the anatomic macula, which in a case of hemianopsia lies at the edge of the seeing retina. This so-called pseudomacula therefore occupies a relatively peripheral position and has taken over the functions of the true macula. Careful investigations have shown that the acuity of vision diminishes from this new center toward the periphery. The acuity of vision of this pseudofovea has been shown to be from one-sixth to one-half better than that of the anatomic fovea in these patients. The same thing applies to colors. The estimate of position of the various points in the retina has undergone an alteration. Stimuli reaching the pseudofovea appear to be straight forward; the localization of other points of the seeing retina have also been changed relatively. This means that the functional area of every part of the calcarine cortex has been changed. In order that this change may take place, the globes must be rotated slightly

toward the side. One calcarine area represents the entire visual center, in spite of the destruction of the other calcarine area, and the brain functions as before, as far as the field of vision is concerned. Fuchs has noted that in cases of a newly acquired hemianopsia, some patients voluntarily turn their eyes somewhat toward the side. In most instances, this probably takes place automatically, the motive being to see objects lying directly in front of us most clearly. In order to do this the object must lie within the field of vision.

The development of a pseudomacula does not take place in amblyopia but only in a complete hemianopsia. As long as there is a substratum which will carry out the old function more or less perfectly, old structures will function in the original manner. It is only when the function is entirely destroyed that the remaining portion of the apparatus will take over the new function. This restitution holds true not only in hemianopsia, but appears to be a general law that applies to all functions. Goldstein has demonstrated this clearly in cases of agnosia in which it is seen that recovery is more complete when there has been a total agnosia than when this has been partial. In cases of total disability, the entire brain seems to make an effort to bring about restitution, which is not true in partial lesions.

WOLTMAN, Rochester, Minn.

A CONTRIBUTION TO THE KNOWLEDGE OF THE TRIGEMINAL NERVE. DE LORENTE, Libro en honor de S. Ramón y Cajal **2**:13-20, 1922.

This is a study of the locus coeruleus and the mesencephalic root of the trigeminus based on Golgi slides of the mouse. Observations on the tract of Probst and the salivatory nuclei are also included.

According to Lorente, some of the afferent axons from the neurons in the gasserian ganglion cross the main sensory nucleus and, after giving off collaterals, travel toward the motor accessory (mesencephalic) nucleus. Before reaching the latter, they divide into an ascending process, which joins the mesencephalic root, and a descending process ending in the main motor nucleus. In some cases, the afferent axons pass directly into the medial nucleus of the locus coeruleus, where they form loose terminal arborizations.

The collaterals described by Willens as arising from the axons of the neurons of the motor nucleus are really axons of association neurons placed in this nucleus, which running toward the dorsal region undergo a bifurcation at the level of the mesencephalic nucleus, one of the processes joining the mesencephalic root while the other joins the tract of Probst, thus establishing connections between the masticatory and salivatory systems.

Two accessory trigeminal nuclei, the intertrigeminal and the supratrigeminal nuclei, are described. The former is placed in the bundles of motor fibers, between the main sensory and the motor nucleus. Lorente believes that this nucleus might well belong to some of the ascending bundles in the field of Monakow or to the lateral lemniscus. The supratrigeminal nucleus is placed over the dorsolateral portion of the motor nucleus, near the main sensory nucleus. It is suggested that this nucleus, which receives collaterals from the fibers of the mesencephalic root, the sensory radiation reaching the locus coeruleus and the secondary pathway of the trigeminus, represents the proximal end of the column of salivatory nuclei of Yagita.

The tract of Probst consists of axons and collaterals from the mesencephalic root. It sends bundles of collaterals, ventrally directed and ending in rich arborizations, thus forming a vertical column of plexuses. This column extends

from the supratrigeminal nucleus to the entrance of the hindmost fibers of the vagus into the tractus solitarius, but it shows its maximum development at the level of the salivatory nuclei.

The axons from the cells in the salivatory nuclei follow varied courses. They form a small nerve leaving the medulla between the motor root of the trigeminus and the facial; in other cases, this nerve leaves the medulla below the exit of the facial. A few cells send their neuraxons to the knee of the facial nerve. Most of the fibers are direct.

Lorente's observations favor Cajal's interpretation of the accessory motor (mesencephalic) nucleus as a center for regulation or control of mastication, since its axons produce a close plexus around the cells in the masticator nucleus. Through the tract of Probst, impulses are also sent to the salivatory nuclei, thus insuring simultaneous mastication and insalivation.

NONIDEZ, New York.

THE RATE OF PRECIPITATION OF RED BLOOD CORPUSCLES IN CASES OF INSANITY, ESPECIALLY IN SCHIZOPHRENIA. A. GLAUS and J. ZUTT, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:66-81, 1923.

The authors state that increased rapidity of precipitation of red blood corpuscles in citrated plasma is due to a decreased stability of the proteins of the plasma. This physicochemical change is a symptom of metabolic disturbances. Increased rapidity of precipitation has heretofore been demonstrated in general paralysis and in many acute and chronic infectious diseases. The authors describe their own technic and discuss the difficulties of obtaining accurate comparable results. They conclude that the rate of precipitation is generally increased in catatonic states but emphasize the fact that schizophrenia in general is not a disease entity. Therefore each case must be considered individually. Cases that are of toxic origin or that are complicated with toxic factors show a rather marked increase of the rate of precipitation when compared with normal citrated plasma.

THE USE OF HYPNOTISM AND THE EMPLOYMENT OF MEDIUMS BY COURTS. HANS W. GRUHLE, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:82-92, 1923.

The possibility of hypnotizing persons against their will and using such subjects for criminal purposes is emphatically denied by the author. The interesting visions of the medium, who apparently gave the state's attorney a fairly accurate description of a murder committed near Heidelberg in 1921 are then described. The information about the crime was given before the mystery had been solved. The author arrives at the conclusion that some consideration ought therefore to be given to the investigation of such bona fide mediums.

SCHIZOTHYM-CYCLOTHYM. HERMAN HOFFMAN, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:93-104, 1923.

The article gives a rather minute analysis of some of the hereditary factors of schizophrenia in the ascending as well as descending branches of certain families. Besides this the changes in personality from schizothym to cyclothym and vice versa found in persons who have inherited both psychic strains from their ancestors are analyzed. There is a more or less successful attempt made to correlate these factors with the well-known laws of inheritance.

NEYMANN, Chicago.

THE ONTOGENETIC DEVELOPMENT OF THE STRIATE BODY OF BIRDS AND A COMPARISON WITH THAT OF MAMMALS AND OF MAN. C. U. ARIENS KAPPERS, Schweiz. Arch. f. Neurol. u. Psychiat., Festschrift f. Constantin von Monakow **13**:348-370, 1923.

While the general form of the brain of *Cyclostomes* and *Selaciens* is comparable to that of mammals and of man in that one finds two hemispheres containing lateral ventricles surrounded on all sides by nervous structures, the telencephalon of bony fish and *Ganoides* have these ventricles surrounded by important structures only on the bottom and the side.

The epistriatum of fish is primarily olfactory. The hyperstriatum is well developed and is divided into superior and inferior parts. The superior part comprises the dorsal and lateral sections of the striate body. It projects from the pallium from which it develops. The hyperstriatum inferior extends farther back than does the former and forms a protrusion into the occipital portion of the ventricles. It is a triangular mass made up of large cells and is rich in medullated fibers. The ectostriatum, like part of the inferior hyperstriatum, is separated from the mesostriatum by the dorsal medullary lamina, better called external medullary lamina, since it surrounds the mesostriatum not only on its dorsal surface but also on its lateral surface. In most of the birds examined by the writer, this lamina is rich in blood vessels which pass to the hyperstriatum. The mesostriatum is usually separated from the hyperstriatum by a deep fissure on the ventricular surface. In larger birds, the mesostriatum may be separated from the hyperstriatum by stripping. The mesostriatum has a caudal prolongation, pars paleostriatica caudata; like the hyperstriatum, however, this is not extensive. In the center of the mesostriatum one finds the basal nucleus, the primitive part of the paleostriatum characterized by the presence of large cells. In the fifteen day chick embryo, on section through the foramen of Monro, one finds the striatum represented on the lateral wall of the ventricle by four prominences, the lower two representing the mesostriatum, the one above the hyperstriatum inferius and the highest one the hyperstriatum superius. The primitive paleostriatum of the chick is augmented by a projection, the mesostriatum, which forms at the base of the brain; on it is superimposed the hyperstriatum, which consists of two parts, the inferior, taking origin from the basal postolfactory area and from the lateral pallium, and the superior, taking its origin from the pallium alone.

The connections of the striate body form a difficult field of investigation. Much was done by Edinger, Wallenberg and Schroeder; the neostriatum is a sensory center which receives fibers from the thalamus through the tractus thalamo-frontalis externus, the tractus thalamo-frontalis medius, and the tractus quinto-frontalis. There are a great many interstriatal connections. The mesostriatum receives frontopetal and frontofugal fibers, the tractus strio-mesencephalicus and the tractus strio-thalamicus internus. The neostriatum is a correlating sensory structure, the paleostriatum an efferent sensory structure. In the neostriatum, we can make only intrinsic divisions; the division into putamen and caudate nucleus is due only to the position of the internal capsule.

In conclusion, it may be said that the striate body of birds, mammals and man is composed of three principal parts—the paleostriatum, the archistriatum (nucleus amygdalae) and the neostriatum. The paleostriatum is derived entirely from the anterior portion of the base of the brain in front of the optic recess. It is represented in amphibians only by the basal nucleus (Palaeostriatum primitif); it becomes considerably larger in reptiles, birds and mammals, forming the paleostriatum augmente, or mesostriatum. The neostriatum takes its

origin partially from the pallium and partially from the telencephalon anterior to the paleostriatum. The projection of this structure toward the rear covers the paleostriatum, the result of a neurobiotactic process, the neostriatum forming connections with the paleostriatum and receiving many striopetal fibers from the thalamus.

WOLTMAN, Rochester, Minn.

FORMATION OF THE CONVOLUTIONS OF THE BRAIN. E. LANDAU, J. f. Psychol. u. Neurol. **30**:201, 1924.

Landau is inclined to agree with Schaffer that the formation of the cerebral convolutions is a primary active process which begins in the sulci and not in the convolutions, but he does not believe that the transitory superficial granular layer plays an active mechanical rôle in the process. This opinion is expressed by him in his recent text-book "Anatomie des Grosshirns, formanalytische Untersuchungen, 1923." It seems that since the publication of this book, Bielschowsky has also discussed this subject (J. f. Psychol. u. Neurol. **30**: Nos. 1 and 2, 1923), and although he quite agrees with Landau's theory, he nevertheless criticizes the arguments adducted by the latter in support of it. According to Bielschowsky, Landau's illustrations are misleading, because, owing to poor fixation methods, they contain many artefacts. In the present paper, Landau meets, and apparently with success, Bielschowsky's criticisms. A reexamination of several of the specimens from fetal brains (4 and 5 months old) has made one point clear, and that is that the status verrucosus found in most of the brains examined has no direct effect on the formation of the convolutions—the verruci are neither the precursors of the gyri nor of the sulci. Bielschowsky, on the other hand, believes that the status verrucosus is not the exclusive cause of convolutional formation. Landau was unable to find in his specimens evidences of primitive gyri or of any precursors of the latter, on which Bielschowsky lays so much stress in his theory of convolutional formation. All specimens, however, showed recesses, smaller and larger, deeper and superficial ones, and it is from these recesses that Landau believes the convolutions are formed. He reproduces in this paper some of his original illustrations, which, he says, every unbiased investigator must admit fail to show the slightest evidence of a proliferating focus in the lamina corticalis. He is firmly convinced that these recesses are not artefacts due to poor fixation, but are the genuine sites of origin of convolutional formation, the precise mechanism of which he promises to publish in the near future.

KESCHNER, New York.

A HISTOPATHOLOGICAL STUDY OF MULTIPLE SCLEROSIS. G. GUILLAIN and I. BERTRAND, Ann. de méd. **15**:476 (June) 1924.

The typical lesion of multiple sclerosis is the plaque, the characteristics of which are well known. The authors note that the subependymal gray matter bordering the ventricles is a frequent site of plaques; the lesions are so extensive at this site that the ependyma is sometimes bordered by a long band almost entirely deprived of myelin. This special type of lesion has received little attention from histologists.

The authors have observed not only typical fully developed plaques, but also plaques in the process of formation. Such plaques are invaded by products

of degeneration, often to such an extent that they present a spongy appearance; a series of cavities of ill-defined contour communicating with each other, contain numerous macrophages carrying lipoid substances in various stages of degeneration. The walls of these cavities are formed by glial fibers entirely deprived of myelin; outside of these larger areas of degeneration are found myelophages and gitter cells. The blood vessels in the areas of intense myelin degeneration present various changes according to the stage and intensity of the process.

A careful histologic technic shows extremely diffuse degenerative changes in the entire spinal cord which are most intense in the immediate vicinity of the plaques. Myeloblasts and myelophages, agents of phagocytosis in situ, are the first elements to appear. Occasionally gitter cells are present. The nerve fibers are involved early. Sometimes there is an edema separating the fibrils, sometimes lateral bulbous formations, destroying a few marginal fibrils. These correspond to degenerative changes in the myelin. No signs of any regeneration of the fibrils have been observed with any method of staining. Myelophages and myeloblasts early invade the nerve fiber acting as phagocytes for the degenerating myelin tissue. The blood vessels even outside the plaques may show slight perivascular infiltration with lymphocytes and with granular bodies of various origin.

DAVIS, New York.

PHENOMENOLOGY. LUDWIG BINSWANGER, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:10-45, 1923.

This contribution gives an extensive description of the theories and facts of phenomenology and a comparison of this method of study with the methods used in the natural sciences, psychology, psychopathology, and psychanalysis. The entire article is a philosophical dissertation on the relation of the observer to the facts observed in the outer world. The author draws attention to the value of comprehending the true essence of apperception rather than the mutation of such apperceptions into personal conclusions by way of the associated ideational processes. These theories are elaborated by the explanation of certain common schizophrenic utterings through the phenomenolistic method. He also explains certain theories of the plastic and descriptive arts by means of this method.

AFFECT AND COLORS, ESPECIALLY FEAR AND BRIGHT-DARK VISIONS. HANS CHRISTOFFEL, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:46-52, 1923.

A description is given of several cases of fear combined with bright-dark visions. The author concludes that definite primary relations exist between fear affect and optical bright-dark visions; these are probably based on peripheral functional disturbances of the visual apparatus. He also states that color symbolism which has thus far been neglected probably plays an important part in psychanalysis.

NEUROSES OF DISPLEASURE AND SPITE, CLEPTOMANIA. LUDWIG FRANK, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:60-65, 1923.

This is a discussion of the early psychogenic causes of cleptomania and a differentiation of such cases from those showing only ethical defects.

NEYMANN, Chicago.

THE TREATMENT OF SEPTIC MENINGITIS BY CONTINUOUS DRAINAGE. WARREN R. RAINEY and LELAND B. ALFORD, J. A. M. A. **81**:1516 (Nov. 3) 1923.

Two cases of recovery from a septic meningitis treated by continuous spinal drainage are reported. Both patients recovered from the meningitis, but one died later, after developing definite signs of pneumonia. At postmortem examination the arachnoid over the cerebral hemisphere was slightly thickened, but without evidence of an acute inflammation.

As contrasted with serum therapy, we believe, on theoretical groups, that drainage offers the better prospect of success. To be successful, drainage should be applied to the lumbar region, should be continuous for several days, and should be instituted early in the infection.

POSTINFANTILE TETANY: REPORT OF THREE CASES. E. T. GIBSON, J. A. M. A. **81**:1687 (Nov. 17) 1923.

Three cases of postinfantile tetany are reported. One was recognized at once, one had been called hysteria before the author saw the patient, and the third was at first considered to be a case of acroparesthesia.

While the peculiar spasm of the muscles of the extremities is the best known manifestation of tetany, one of the cases reported shows that effects due to involuntary and sensory nerves (dyspnea, diarrhea and atrophy of hair) may be just as prominent. Occasionally paresthesias of the extremities without organic neurologic signs are due to tetany. In one case, there was a typical attack of tetany four months before thyroidectomy; it is possible that it was a case of essenial hypoparathyroidism.

Treatment with calcium was satisfactory, so far as symptoms were concerned.

NIXON, San Francisco.

THE OLFACTORY SENSE OF THE CABBAGE BUTTERFLY, PIERIS RAPAE LINN., AN EXPERIMENTAL STUDY. DWIGHT ELMER MINNICH, J. Exper. Zool. **39**:339 (April 5) 1924.

In insects, feeding, sex attraction, oviposition, and social habits are all dependent to a large extent on the olfactory sense. The location of the sense organs has remained a subject of controversy. Using the extension of the proboscis as an index of olfactory stimulation, the author attempted to determine the location of the olfactory organs in the cabbage butterfly, *Pieris rapae Linn.* The butterflies were suspended in an odor chamber by their wings and the odorous material, which was a commercial preparation of apple juice, was placed on the bottom of the chamber. The labial palps were removed to expose the proboscis coil. A trial lasted one minute, and a failure to observe any extension of the proboscis during this interval was recorded as "no response." The antennae were eliminated by coating them with petrolatum, by covering them with a mixture of paraffin and petrolatum, or by amputating them with fine scissors. When one antenna is eliminated from possible olfactory stimulation, the olfactory response is reduced very little, on the average 6 per cent. When both antennae are eliminated, the olfactory response is reduced greatly, on the average 58 per cent. These decreases of response are due primarily to the elimination of olfactory organs, and not to a generally abnormal state of behavior following operation. In *Pieris*, the antennae probably constitute the most important olfactory area, each

antenna being richly equipped with olfactory organs. The antennae do not constitute the sole olfactory area in *Pieris*. In extremely sensitive animals, mechanical stimulation may also effect proboscis extension.

WYMAN, Boston.

THE ACTION OF CRATAEGUS OXYCANTHA ON THE TONUS OF THE INVOLUNTARY NERVOUS SYSTEM. SANTENOISE and VIDACOVITCH, L'Encephale **19**:246 (April) 1924.

Crataegus Oxycantha (a drug obtained from the English hawthorne, the French name being aubepine) is reported by these authors as being a valuable depressant of the sympathetic division of the vegetative nervous system. From clinical observations, it is claimed that administration of this drug causes a disappearance of symptoms due to sympathetic overactivity, a reduction of the solar plexus reflex and an increase in intensity of the oculocardiac reflex. The authors use the tincture in doses of from 20 to 60 minims (1.25 to 4 c.c.) per day.

IRREGULARITIES OF RESPIRATORY RHYTHM. G. LEVY and M. L. VAN BOGAERT, L'Encephale **19**:302 (May) 1924.

In a brief paper which is illustrated by tracings of respiratory curves, the authors point out that variations in the rate and depth of the respiratory rhythm occur especially in syndromes in which there is involvement of the extrapyramidal pathways. In only one of a number of patients with true bulbar involvement were similar phenomena observed. The authors believe that these respiratory phenomena are due to the same cause which is responsible for the hypertonia and tremor observed in basal ganglia disease.

The authors refer to the work of Parisot in 1902 and of Klippel and Boeteau in 1892.

HYSLOP, New York.

THE TREATMENT OF SADO-MASOCHISM. G. STROEMME, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:354-361, 1923.

The writer reviews the psychanalysis of five cases of sadism and masochism. The patients' complexes were closely related to their internal dislike for work. During the analysis, masturbation was not forbidden; rather encouraged. This is a safety valve for the patient, which gives him an opportunity to act out his phantasies for the time being.

EXPERIMENTAL INVESTIGATIONS OF THE ATTENTION AND COMPREHENSION OF THE INSANE. ERNEST WENGER, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:362-384, 1923.

The psychologic method of Trueb for measuring attention and comprehension was used. Many psychotic patients were examined, and the results were tabulated and compared with those obtained in normal persons. The procedure was found to be quite useful, and the following conclusions were made: Senile persons cannot increase their comprehension by increasing their attention. In organic psychoses, improvement can be shown by this method whenever the patients are in the process of recovering their faculties. Mixed symptoms give results similar to those obtained by other methods of investigation.

NEYMANN, Chicago.

MULTIPLE NEURITIS FOLLOWING CARBON MONOXID POISONING: A CLINICO-PATHOLOGIC STUDY. GEORGE WILSON and N. W. WINKLEMAN, J. A. M. A. **82**:1407 (May 3) 1924.

Three cases are reported, two with necropsy, of patients who had evidence of polyneuritis occurring as a sequel to gas poisoning. These patients had evidence of multiple neuritis, although the picture was unusual in that the deep reflexes were increased. The authors believe this increase in the deep reflexes is probably due to an involvement of the globus pallidus or of the cortex or of both. In one of the cases a paradoxic condition was found in that there was spasticity and increase of the deep reflexes at the knees, while at the ankles flaccidity and lost reflexes were found.

Peripheral nerves stained by the Marchi method showed a marked increase of Elzholz bodies, which tended to localize, leaving portions of the nerve fiber free. The medullary sheath was swollen and tumefied. Rarely was there axis cylinder change, and when present it consisted of a fusiform swelling.

Multiple neuritis occurring as a sequel to carbon monoxid poison is probably more frequent than is commonly believed, yet direct pathologic evidence of such an involvement is practically unknown in the literature. The peripheral nerves are not often removed at necropsy, and this is probably the reason why changes in these parts are not more frequently discussed.

NIXON, San Francisco.

ALCOHOLIC INSANITY (ACUTE ALCOHOLIC HALLUCINOSIS) AND ITS RELATION TO SCHIZOPHRENIA. MAX WOLFENSBERGER, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:385-420, 1923.

Sixteen cases of alcoholic hallucinosis were carefully investigated. Fourteen of these were in men and two in women. The ages ranged between 32 and 60 years. In 75 per cent. of the cases a psychotic ancestry was demonstrated. The affect was mostly one of fear mixed with depression; in one or two cases it was one of fear and euphoria. Ten of these patients, eight men and two women, later developed outspoken schizophrenia, mostly with deep dementia. Two other male patients developed a psychopathic condition with schizophrenic paranoid symptoms. One patient was at least somewhat schizophrenic during his stay in the institution. The final outcome in this case could not be ascertained because the patient disappeared from observation. Only a single patient showed no psychotic trends after he had recovered from the acute alcoholic hallucinosis. This demonstrates, according to the author, that by far the greater number of cases of acute alcoholic hallucinosis can be looked on as a symptom picture, the reaction of a schizophrenic brain toward a definite kind of alcoholic intoxication.

NEYMANN, Chicago.

MENINGITIS: DISTRIBUTION ACCORDING TO AGE AND ETIOLOGY. JOSEPHINE B. NEAL, J. A. M. A. **82**:1429 (May 3) 1924.

In a list containing 1,535 cases of meningitis, arranged according to age and etiology, it is seen that with the exception of tuberculous meningitis, more cases of meningitis occur in the first year of life than in any other one year. The number of cases of meningococcic meningitis in the first year of life far exceeds those in any other year. The greatest number of cases of tuberculous meningitis is found in the second year of life.

As to the frequency of the various types, the author notes that in times when there is no epidemic, the number of cases of tuberculous meningitis equals or exceeds the number of cases of meningococcic meningitis. After the meningococcus, the pneumococcus and the streptococcus are the most common causes of purulent meningitis, followed by the influenza bacillus, the staphylococcus and *Bacillus coli*, in the order named.

NIXON, San Francisco.

TREATMENT OF GASTRIC NEUROSES. JACOB KLAESI, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:122-130, 1923.

A starvation diet for hysterical patients followed by the foods which caused them the most trouble is recommended. (This would seem to be a rather heroic procedure.)

CONSTITUTION AND RACE. ERNST KRETSCHMER, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:138-147, 1923.

The author discusses the differences in psychic and physical constitutions of the two main races of Germany, the Nordic and the Alpine. He classifies the former as mainly schizothym in makeup, the latter as mainly cyclothym.

NEYMANN, Chicago.

Book Reviews

CONCERNING SOME HEADACHES AND EYE DISORDERS OF NASAL ORIGIN. By GREENFIELD SLUDER, M.D. Pp. 260 with 115 illustrations. St. Louis: C. V. Mosby Company, 1920.

This is an interesting and timely book, which is of especial interest to neurologists because it discusses with a great deal of clearness certain disputed syndromes, notably the syndrome of nasal ganglion neuroses or sphenopalatine neuralgia. There is an excellent introduction to the general pathology and anatomy by Jonathan Wright, which should be of interest chiefly to those specializing in the treatment of diseases of the nose and throat. It shows clearly the pathologic process which brings about an occlusion, and thereby a gradual pressure on certain bony canals, with a consequent involvement of the nerve trunks.

Chapter one concerns itself with vacuum frontal headaches, with eye symptoms only. This subject is rather new to neurologists. Sluder defines it as a low-grade, unending headache, resulting from closure of the frontal sinus, without nasal symptoms or signs, and made worse by use of the eyes. These patients have ocular symptoms only. The headache is usually frontal and is rarely referred to the external angular process of the frontal bone. The nasal trouble in these cases is revealed by tenderness of the upper inner angle of the orbit, this being the point at which the frontal sinus is thinnest. Pain on pressure at this point, which is known as Ewing's sign, is sometimes the *only* indication of the nasal trouble, the rhinologist's examination being negative. The etiology consists in the closure of the frontal sinus outlet.

The second chapter discusses sphenopalatine neuralgia. As is well known, the author described this syndrome years ago, and his original conception has been greatly enlarged by excellent work on his part. This so-called lower type of facial pain is different from the usual pains resulting from fifth nerve disease. Many competent surgeons and neurologists do not believe in the existence of Sluder's syndrome, and believe that the pain which he describes is merely the spilling over of pain from fifth nerve disease. One argument which neurosurgeons constantly hold out against Sluder's idea is that in most instances alcoholic injection into Meckel's ganglion does not bring about a cessation of the pain.

In the present volume, he explains this by the fact that the pain is the result of the involvement of the maxillary and Vidian nerves, central to the ganglion, secondary to sphenoidal inflammation, and he believes that intrasphenoid application of cocaine will stop such pain. This may be an explanation of the hitherto disputed point.

Chapter three takes up hyperplastic sphenoiditis and its enviroining nerves —optic, oculomotor, trochlear, trigeminal, abducens, Vidian nerves and nasal ganglion. This chapter is interestingly written. There is an excellent introduction, giving the anatomic relations in which the method of involvement of the different cranial nerves is clearly shown. The author discusses at length so-called migraine, and states that hyperplastic sphenoiditis inflammations are a frequent cause.

In discussing hyperplastic bone processes he states that the maxillary and Vidian nerves are most frequently infected, next in order the optic nerve, and

that the oculomotor, trochlear, ophthalamic and abducens are less frequently involved, because the latter nerves are located in the soft confines of the sphenoidal fissure.

From the foregoing it can readily be seen that the subjects discussed are of extreme interest to the neurologist. The causes of headaches, optic atrophy and pains in the face frequently baffle physicians. Dr. Sluder's book undoubtedly contributes much to the elucidation of the causes of these phenomena. The book is well prepared, the illustrations are excellent, and the publishers have done their work well.

ZUR KLINIK DER NICHT PARALYTISCHEN LUES-PSYCHOSEN (NONPARALYTIC SYPHILITIC PSYCHOSES). By DR. H. FABRITIUS. Pp. 103. Berlin: S. Karger, 1924.

This is a well-considered essay on the classification and symptomatology of syphilitic psychoses, or of psychoses in syphilitic patients, general paralysis excluded. Evidently the work was produced under the influence of Bonhoeffer, and reflects, or contains, much of his calm judgment and common sense. Of the twenty-three carefully observed and fully reported cases, twenty-two are from his service at the Charité in Berlin.

The German literature is fully and carefully considered; otherwise the bibliography is incomplete to the extent of deformity. Of the forty-two references, thirty-seven are German, two Scandinavian, two French and one Dutch.

After twenty-one pages of mature consideration of opinions and reports, the author makes the following classifications:

1. Exogenous reaction types with acute or protracted course.
(*a*) Anxious-confusional states of excitement.
(*b*) Epileptogenic cloudy states.
2. Hallucinosis or hallucinatory paranoid cases.
3. Chronic organic psychoses.

To these he adds manic, melancholic, catatonic and other psychotic states in syphilitic persons.

The case reports (seventy-six pages) present examples of all of these types.

The monograph is an excellent and well balanced summary of the subject, and the cases are a real contribution.

Obviously, numbers of problems connected with the occurrence of mental disorders in persons infected with syphilis remain unresolved. How much has the syphilitic infection per se, how much have the syphilitic lesions of the brain, to do with a psychosis appearing in the infected person? Just what is the import of the topography of the lesions? What is the rôle of temperament or mental trend of the patient, and how important is emotional stress or shock in the genesis of such a psychosis? These questions are soberly considered, but they have not been answered.

AN INTRODUCTION TO THE STUDY ON MENTAL DISORDERS. By FRANCIS M. BARNES, JR., St. Louis: C. V. Mosby Company, 1923.

This is a sound book to put into the hands of medical students. The hundred odd pages dealing directly with the different mental diseases and forming the second part of the book are merely brief lecture notes. The larger first part has in it more of interest. The discussion of methods of study is well balanced but seems rather short. Longer and interesting chapters discuss psychology and medical practice, observation of mental cases, and treatment.

The psychiatric social worker is given considerable attention. "A social worker with but a special training." Incidentally, the author abuses the word "but." He makes up for this by a discussion of how the social worker can get help from the psychiatrist in many cases where there is no frank mental disease. His common sense again appears when he says, that "the psychiatrist is not a performer of miracles" and that the social worker is not to expect too much.

The short chapters on psychologic functions and their common disturbances are satisfactory. Other chapters reflect the author's interest in vocational guidance, evidently studied in connection with Veterans' Bureau work, and the bibliographies especially reflect a certain absorption in the productions of one very good locality, St. Louis.